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FIFTY-THIRD ANNUAL MEETING

Harry E. Ungerleider, M. D.
Editor

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Transactions of the Association of Life Insurance Medical Directors of America

FIFTY-THIRD ANNUAL MEETING

The Fifty-Third Annual Meeting of The Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania in New York City on Wednesday and Thursday, October 21 and 22, 1942.

PRESIDENT WENSTRAND—In calling to order this fifty-third meeting of our Association, once more I wish to express my Company's and my sincere appreciation for having been elected to act as your President. I hope that what has been done during my term of office has fulfilled your expectations. Mine has really been a minor part. Most of the credit is due those who have worked with me. I have in mind especially our efficient Secretary, Dr. Dewis, who has also served on the Program Committee with Dr. Ungerleider, the versatile Editor of our Transactions, as well as all committee chairmen and members. In the presence of this assembly I thank you one and all most heartily.

It is not for me to dwell upon the present emergency; that falls to the lot of others, but I am satisfied that our meeting is warranted. We are in a measure doing our part when we consider problems which deal with the security of future generations. The more than one hundred twenty-four billion dollars of life insurance in force is a backlog which cannot be ignored. It is our job not only to continue as we have but also to improve medical risk selection. I am confident that

our speakers and their subjects will add to and refresh our knowledge.

For a few minutes I shall turn back the pages of history to show you some pictures of our past.

Just after The Specialized Mortality Investigation had come from the press my first steps in medical risk selection were guided by one of the earliest members of our Association, and, having continued with him for a third of a century, perhaps I may be forgiven in speaking particularly of this man and his influence upon our development.

Dr. Fisher was elected to membership at the first regular meeting in 1890 when he was Assistant Medical Director of The Northwestern Mutual Life Insurance Company and at once became an enthusiastic worker in the organization. Two years later he was chosen Vice-President. Some incidents to which I shall refer certain of you may have heard before, but as recently reminiscent notes have been found, apparently dictated by the Doctor at various times though not dated, it seems pertinent to quote from these quite freely.

The story which follows in his own words I have heard him tell many times.

"After graduating from Rush Medical College, I served as intern in Cook County Hospital. When my internship had been completed, I was appointed Assistant Medical Superintendent of the Wisconsin State Hospital for Insane at Mendota. While holding this position, I attended meetings of the National Association of Medical Superintendents for the Insane. At these gatherings I derived most valuable information and at a time when most needed.

"In August, 1881, I resigned and engaged in private practice in Milwaukee, Wisconsin; served as local examiner for several life insurance companies, and in 1884 was appointed local examiner for the Northwestern Mutual. In December, 1885, I became Assistant Medical Director, giving only a portion of my time to details in the Medical Department during the following three years. In 1888, owing to the increase in work

of the Medical Department, I abandoned my private practice to give full time to the Company.

"While in practice, I was a member of the Local and State Medical Societies and was at a loss to understand why the medical directors of life insurance companies failed to avail themselves of the value of a similar organization. I discussed the subject with my superior, Dr. Lewis McKnight. He was of the opinion that, owing to the great rivalry then existing between life insurance companies, it would be difficult to convince the medical directors that such an organization would be feasible. I asked if there had ever been an effort made in that direction. He answered, none to his knowledge.

"When Company business in October, 1888, necessitated a visit to New York, I decided to call on the Medical Directors of the Equitable Life, the New York Life and the Mutual Life. Realizing the struggle going on among the so-called "big three", each striving to become the largest, it seemed best to me to secure the views of these doctors on the value to be derived from an organization of life insurance medical directors. I met Dr. Lambert of the Equitable, Dr. Winston of the Mutual Life and Dr. Huntington of the New York Life. None of them appeared to have given any serious consideration to the subject. Dr. Huntington seemed somewhat more interested than the other two and it was he who a few months later suggested to Dr. Keating, Medical Director of the Penn Mutual, to call a meeting of medical directors, who met May 29, 1889, at the Union League Club in New York.

"At this meeting a committee was appointed to consider the advisability of establishing a permanent organization. This committee was ready to report in December, 1889, at which time a constitution drafted by it was adopted. The first regular meeting was held at the Buckingham Hotel, New York City, on May 30, 1890.

"The Actuarial Society of America came into being at practically the same time, so the year 1889 marked the birth of insurance medicine and scientific life insurance risk selection.

Prior thereto risks were almost entirely selected according to the medical experience and the personal judgment of the medical director.

"Our Association has prospered and been of value as our genial friend Dr. Willard so well said at its thirty-fourth annual meeting: 'The culminating point in the education of a medical director comes when he has the opportunity of joining The Association of Life Insurance Medical Directors. He will be a better director, a better doctor and better serve his Company after he has joined this organization, which radiates friendship and shares the knowledge possessed by all. It is the clearinghouse of information for the benefit of all who are engaged in this business and stimulates inquiry and research, which as time goes on will be more and more important.'"

In the Proceedings of this meeting just referred to is found a memorandum written by Dr. Fisher dealing with the background of the Specialized Mortality Investigation. One paragraph reads as follows: "At the third meeting in 1892, having in mind the law of general averages, which from an insurance standpoint is well-defined and reliable in large groups carried over a long term of years, I was led to introduce a resolution inviting member companies to enter into a scheme of accumulating records covering certain medical impairments, including a family record of tuberculosis, with a view of combining such data on insured lives, and after a sufficient time had elapsed to determine the mortality on each class as an aid in risk selection. This was the first attempt in this country to consider the question of collecting the mortality of American life insurance companies with reference to special classes of risks."

The introducer of the resolution was made chairman of the committee which was instructed to carry on. Just how soon the companies began to write these so-called class cards covering twelve impairments is not definitely known, but at least one card was saved by John Gallagher, who became Dr. Fisher's errand boy in 1889 and who is still on the job. This card

bears the date June 9, 1893. John has also saved other valuable historical material, including the circular letters dated May 11, 1894, which were written to twenty-seven companies and with space below to report progress. Twenty-two answered, but the number of cards then on hand was woefully small.

The 1893 meeting was held June 6th at the Home Office of the Northwestern Mutual in Milwaukee, no doubt because then the Chicago World's Columbian Exposition was in progress. Only sixteen members were present. It was important only because Doctors O. H. Rogers, E. K. Root and T. H. Willard, among others, were elected to membership. What they did for our Association is well known and especially Dr. Rogers has left an indelible record of accomplishment never to be forgotten.

The early meetings were addressed by eloquent and scholarly men such as Doctors Holden, Hamill, Russell, J. L. Davis, Homans, Root, Burrage, Grant and Lambert. Their papers were long and philosophical; most were literary compositions of real merit. Outstanding subjects were, albuminuria, heredity, rheumatism, gout, appendicitis and medical examiners. To begin with, the essayists were selected in open meeting a year in advance and topics for general discussion assigned. At the ninth meeting the subject was "Sugar in the Urine". During the tenth, Dr. Shepherd and Dr. Fisher led a discussion on "Build, Height and Weight — Its Bearing upon Longevity", and Dr. Rogers presented a standard graphic table of heights and weights prepared from New York Life data. The titles of some papers were rather unique. For example, "Influence on Longevity of the Use of Large Quantities of Beer", and "The Relation of Surgical Mutilations to Longevity." It interested me very much to find that Dr. Root of the Aetna in the 1899 meeting gave an illuminating essay on "Differential Diagnosis of Cardiovascular Murmurs" which he described in the following words: "Functional heart murmurs of a variety frequently met with by life insurance examiners, but more often overlooked or

misinterpreted by physicians examining patients ill with some disease." Yet medical departments did not begin to differentiate and select non-organic heart murmurs until much later. I recall that when I began to make examinations in the Home Office of The Northwestern Mutual all murmurs caused rejection before 1907.

The beginning of taking blood pressure readings in life insurance examinations has been covered many times, but in one of Dr. Fisher's memoranda is a significant paragraph telling of an incident which as far as I know has not appeared before. In our Transactions the sphygmomanometer is first mentioned by Dr. Root in his discussion of Dr. Arthur Pell's paper on heart murmurs at the sixteenth meeting, October, 1905. I quote:

"Prior to the fifteenth annual meeting of the Association of Medical Directors, held at the New York Academy of Medicine on June 2 and 3, 1904, I had not seen a sphygmomanometer. At this meeting one of the members, whose name I do not recall, exhibited a crude spring instrument he had secured abroad. It was not a Gaertner Tonometer. As I remember it was used to measure the pressure of gas, resembling somewhat the present Tycos, but considerably larger. With the use of an arm band or cuff he took the pressure of a number of the members present. Dr. Emery of the Manhattan Life had quite a high pressure. Some of the members refused to submit to the experiment."

Dr. Fisher probably mentioned the Gaertner Tonometer and an arm band because a few months later one of my Chicago friends brought one of these appliances with a finger cuff from Vienna, which I saw and we discussed in our department but found inadequate. It was not until the fall of 1906 that our first Janeway sphygmomanometer was secured and used.

Dr. Fisher worked and lived long enough to hear from his many friends and admirers both in his Company and in this Association words of recognition for his accomplishments. He was a man liberally endowed with an inquiring mind;

"scientific curiosity", as one of his associates called it. He was firm, but when facts warranted did not hesitate to change his mind. He has left a lasting mark in his Department which he ruled for fifty years, and also on this Association together with other outstanding pioneers in modern risk selection.

Dr. T. H. Dickson, Chairman of the Medical Section of the American Life Convention, is here, and I take pleasure in asking him to say a few words to us.

* * * * * *

DR. DICKSON—Mr. Chairman and Gentlemen: It is a very real pleasure to bring greetings from the American Life Convention. Many of you are connected with companies numbered among the hundred and sixty-eight member companies of that organization, and ipso facto are members of the Medical Section. To all of you, whether members or not, it is a pleasure to extend greetings and a most cordial invitation to attend our meetings. Some of you have been with us, either as participants or as essayists, and know that we always have a good time.

When we shall meet and where we shall meet, I cannot say. Your chairman has just told you that the next meeting, the 1943 meeting of this organization, is for the present out, and I think by afternoon, I shall know more definitely when the Medical Section of the American Life Convention will not meet, but when we do meet, wherever we meet, we shall be delighted to have you with us.

PRESIDENT WENSTRAND—We now come to the regular program, and Dr. Earl C. Bonnett, Assistant Medical Director of the Metropolitan Life Insurance Company, together with Mr. Edward A. Lew, Assistant Actuary, have prepared a paper on "A Mortality Study of Systolic Heart Murmurs." Dr. Bonnett!

A MORTALITY STUDY OF SYSTOLIC HEART MURMURS

By Earl C. Bonnett, M. D.
ASSISTANT MEDICAL DIRECTOR

AND

Edward A. Lew
ASSISTANT ACTUARY

Metropolitan Life Insurance Company, New York

There have been so many excellent papers on heart impairments presented to this Association by distinguished authors that it is with some sense of trepidation and apology that we bring the subject again to your attention.

Recently we completed an investigation of the mortality among insured lives presenting various impairments. The investigation was undertaken primarily for our own information, in conformity with a program of periodically testing our underwriting practices. Included in the investigation were several common types of systolic heart murmurs in regard to which we had for some years felt that our underwriting practices could be improved. This paper is confined to a review of the experience under heart murmurs classified as follows:

1—Constant, systolic, apex murmur, not transmitted
2—Inconstant, systolic, apex murmur
3—Constant, systolic apex murmur, transmitted to the left and/or mitral regurgitation
4—Constant, systolic, apex murmur, and/or mitral regurgitation with history of rheumatism, chorea, tonsillitis, or other streptococcic infection.
5—Systolic apex murmur, not falling clearly into categories 1, 2 or 3.
6—Heart murmur without details.

Each of the above classes, except Class 4, included only those cases on which the designated type of heart murmur was the sole rateable condition found; that is, cases with other impairments rated +15 or more, and cases with rateable occupations or with other rateable factors were excluded from the investigation. Class 4 included cases which would have been assigned to Class 3 or to Class 1 (beginning in 1935) except that in addition to the murmur there was also a history of rheumatism, chorea, tonsillitis or other streptococcic infection. Classes 1, 2, 5 and 6 include a few cases with a history of rheumatism or other infectious disease, where the rating for such a history was less than +15.

The investigation was made on the basis of our medico-analytical card records. These cards were prepared shortly after issue by a carefully trained staff of girls working under instructions from the Medical and Actuarial Divisions. The medical impairments were recorded on the cards in the usual code. The coding of each case was primarily based on the information reported by the examining physician, and was largely independent of the medical underwriter's appraisal of the impairment. The investigation covered both the standard and substandard Ordinary issues of 1925 to 1939 inclusive, traced to December 31, 1941. For the purpose of determining the expected deaths, a special select mortality table was constructed representing the Company's experience on standard Ordinary issues of 1925 to 1939 inclusive, traced to December 31, 1941. Accordingly, the ratios of the actual number of policies terminated by death to the number expected by this special mortality table reflect the experience on particular medical impairments in terms of the Company's average mortality on standard Ordinary issues.

Nature of the Data

In considering the mortality experience under heart murmurs subdivided into broad classes on the basis of the information reported in applications for life insurance, it is important to

keep in mind that we are not dealing with homogeneous groups of accurately diagnosed lesions or at times even with closely related clinical entities. The coding of individual cases into one of the six classifications was based primarily on the examining physician's description of the murmur rather than on his remarks by way of diagnosis. The principal reason for the heterogeneity of the heart impairments included under the several classifications seems to lie in the considerable variance of the terminology used to describe heart murmurs. For instance, a review of a large sample of applications included in the investigation indicated that many physicians tend to report their heart murmurs in stereotyped terms, the precise verbal formulae used differing in various localities either because of local customs or differences in medical education. Thus it is entirely possible for a heart murmur, presumably that of mitral regurgitation, to have been reported from Chicago in terms that fitted the definition of one code while the same anatomical lesion reported, say from Baltimore, in somewhat different terms would have fitted into the definition of another code.

The increasingly more careful investigation and underwriting of heart murmurs has also introduced an element of heterogeneity into the picture. Thus, while the Metropolitan began using a heart chart as early as 1926, the form of the chart has since been revised and improved several times. As far back as 1926 our examining physicians were also instructed to take a blood pressure reading in any case where there was doubt as to the significance of a heart murmur. In 1934 our practice was modified so as to require the taking of a blood pressure whenever a heart murmur was found; however, some time elapsed before this rule was generally observed. At no time have urinalyses been routinely requested in cases where a heart murmur is found, but since about 1935, there has been a tendency to obtain urinalyses in questionable heart cases. During the last six or seven years, we have also resorted more frequently to second heart examinations to clear up doubtful descriptions of heart murmurs or

to establish criteria of cardiac fitness. As a result of concurrent blood pressure determinations, urinalyses and the more careful investigations made in recent years, unfavorable heart conditions which might earlier have been passed unnoticed have been brought to light. To this extent the material included in the study, arising from recent issues, definitely represents more carefully selected risks.

Since the classification of heart murmurs has been made largely on the basis of the examining physician's description and under changing underwriting procedures, there is little doubt but that even the most carefully coded group of cases represents heart impairments of varying significance. In the 1920's when investigation of heart murmurs was less rigorous and fewer blood pressures were taken, each broad class probably included a higher proportion of more serious murmurs than in recent years. The codes used for classifying heart murmurs were apparently defined to accommodate the wide variation in the examining physicians' reports and it is our opinion that in spite of the increasing use of heart charts and more careful cardiac investigations, we cannot by our present code definitions set apart the cases which are clinically related or the murmurs which are approximately of the same significance. Accordingly, the mortality experienced in each of the classes here studied furnishes a guide of only limited relevance for cases where specific and accurate diagnoses have been established.

Constant Systolic Apex Murmur Not Transmitted

This type of murmur is quite commonly reported. In our standard and substandard Ordinary issues of 1925–39 it occurred with a frequency of about 39 per 100,000 policies, accounting for nearly 9% of the accepted heart murmurs. Only about 6% of the cases included in this class were rated standard, about 17% were rated from +30 to +50, and about 66% were rated from +55 to +100. Included among the 11% of the cases rated +105 to +150 were a few with a

history of rheumatism or other infectious disease, where the rating for the history was less than +15; beginning in 1935 such cases were included in Class 4. The average rating for all the cases in this class which were rated substandard was about +95.

The murmurs in this class were variously described as slight, soft, blowing, rough, and rasping. In many instances the examining physician gave the diagnosis as "functional" or "insignificant." Some cases diagnosed as "mitral regurgitation" were coded in this class because the murmur was described as not transmitted.

There were no deaths among the relatively few cases issued at standard rates.

The experience on the cases issued at substandard rates is given in Table I.

The mortality ratio of 192% for the issues of 1925–1939 corresponds closely to the average rating (about +95) assigned to the cases included in this experience. However, the very small data on the issues of 1932–1939 show a mortality ratio of only 130%. Elimination from the experience of cases with a history of rheumatism (rated less than +15) reduces the mortality ratio for the issues of 1925–39 to about 185% and that on the issues of 1932–39 to about 126%.

Several years ago Dr. A. Hunter in his paper on "Heart Murmurs" (T. A. S. A. XXXVII, p. 380) gave details of the New York Life's experience on this type of murmur; the experience on the substandard issues of 1915–1924 traced to anniversaries in 1935 showed a mortality ratio of 146% (184 deaths) while the corresponding experience on substandard issues of 1925–1934 also traced to anniversaries in 1935 showed a mortality ratio of only 109% (33 deaths). Another large company's more recent experience under substandard issues with this murmur showed a mortality ratio definitely in the substandard range.

In the Medical Impairment Study, the experience under substandard issues with this murmur showed a mortality ratio

TABLE I

Constant Systolic Apex Murmur Not Transmitted, without Hypertrophy
Experience on Substandard Issues of 1925–1939 Traced to December 31, 1941

	Mortality Ratio			Number of Policies Terminated by Death		
	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939
Experience by age groups at issue:						
Under 40	163%	*( )%	141%	13	8	21
40 and over	381	145	253	22	10	32
All ages	255	130	192	35	18	53
Experience by policy year duration:						
1–5	297	149	198	15	15	30
6 and over	230	*( )	185	20	3	23
All ages	255	130	192	35	18	53

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

of 156% (234 deaths). However, the mortality rates per 1,000 in the Metropolitan experience presented here are almost identical with those in the corresponding Medical Impairment Study experience. In other words, there has apparently been no change in the absolute mortality rates of persons with this type of heart murmur from the period covered by the Medical Impairment Study (i.e., 1909–1928) to the period covered by the Metropolitan experience (i.e., 1925–1941). However, as mentioned before, the Metropolitan's experience on the more recent issues (1932–1939), while quite small, shows lower mortality than that experienced over the entire period covered by the investigation (1925–1941).

In his note on "Functional Heart Murmurs" (T.A.S.A. XXXVIII, p. 141), Dr. A. Hunter gave the experience of four large companies under standard issues with this murmur; this experience covering the issues of 1925–1935 traced to anniversaries in 1936 showed a mortality ratio of 115% (97 deaths). A very small experience on standard issues in the Medical Impairment Study showed a mortality ratio of 93% (16 deaths).

Our underwriting practice with respect to cases where the examining physician reported an apex systolic murmur not transmitted, with qualifying remarks such as mitral regurgitation, was to rate them on the basis of the underwriter's over-all appraisal. Until about 1930 such cases were nevertheless coded as apex systolic murmur not transmitted and it was not until 1935 that mention of mitral regurgitation in connection with an apex systolic murmur not transmitted excluded the case from this class. It also appears that cases of apex systolic murmur not transmitted found on examination but with a record of other more serious heart murmurs were routinely coded as apex systolic murmur not transmitted, even though the record may have been given some weight in arriving at the rating on the case. Practically all such questionable cases with this murmur were apparently given substandard insurance, and their inclusion in this class

with other cases rated substandard because of the Company's rating schedule would go a long way towards producing substandard mortality on substandard issues. On the other hand, the carefully selected cases which in other companies were granted standard insurance have produced mortality in the range from standard to borderline. It is, of course, possible that, even among the cases issued at standard rates, there were some where the examining physician erroneously stated that the murmur was "not transmitted." This would naturally have occurred more frequently in the days of less careful heart examinations and the elimination, by more careful study, of murmurs which were actually transmitted may account for the improvement in the Metropolitan's experience on the substandard issues of 1932–1939. If we assume that the higher mortality experienced on substandard issues reflects the inclusion in the class of a number of more serious murmurs, due to the coding procedures and the less rigorous heart examinations made in the past, it would appear that in carefully selected cases this murmur may safely be taken at standard rates, but that on doubtful cases such as apparently comprised a sizeable proportion of the Metropolitan's substandard experience, substandard mortality is to be expected. Careful selection of cases with this murmur involves separating out the murmurs which though described as "not transmitted" are in reality so low pitched that the transmission is missed, unless the doctor's hearing is especially acute or he examines the applicant after exercise.

In the Medical Impairment Study experience and in the New York Life's experience on substandard issues (cited above) the relative mortality at ages 40 and over at issue was definitely lower than at ages under 40 at issue. In the four large companies' experience under standard issues the relative mortality at ages 40 and over at issue was only slightly lower than at ages under 40 at issue. This mortality trend by age groups at issue is consistent with the hypothesis that a certain proportion of the cases included in this class (larger

on substandard issues than on standard issues) involved more significant murmurs on which the mortality is relatively higher at the younger ages. The Metropolitan's experience on substandard issues presented here indicates, if anything, higher mortality at ages 40 and over at issue, but we believe that this feature of our experience reflects the inclusion of a large number of cases with mitral regurgitation in the age group 40–49 at issue.

As regards the causes of death in the Metropolitan's substandard experience on these cases without hypertrophy there were 25 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45), as against 5½ expected deaths from these causes. This accounts for over 70% of the extra mortality on the substandard cases with this heart murmur. The fact that there were so many extra deaths from heart disease, in itself indicates that there must have been a considerable number of persons with more serious heart diseases included in this class.

There was a small number of cases with the constant systolic apex murmur not transmitted on which some degree of hypertrophy, mostly very slight, was reported. All of these cases were accepted at substandard rates. There were only six deaths equivalent to a mortality ratio of 193% among them.

Inconstant Systolic Apex Murmur

In our standard and substandard Ordinary issues of 1925–1939, this type of murmur occurred with a frequency of about 48 per 100,000 policies. This relative infrequency makes us suspect that many of our examiners do not report it when they consider it insignificant. This, of course, is contrary to their instructions but is usually an effective method of avoiding second examinations, the completion of heart charts and fussy questions from the Home Office. Despite this situation the inconstant systolic apex murmur accounted for over 10%

of the accepted heart murmurs and was the most common of the presumably unimportant murmurs. Over 35% of the cases included in this class were rated standard, about an equal proportion was rated from +30 to +50, and most of the remaining cases were rated from +55 to +100. Only about 2½% of the cases were rated from +105 to +150 and among these were included a few with a history of rheumatism or other infectious disease, where the rating for the history did not exceed +15. The average rating for all of the cases in this class which were rated substandard was +55.

The vast majority of the murmurs falling into this class were inconstant in relation to exercise. Many of them were "brought out by exercise," but some "disappeared on exercise," some were described as "not heard in recumbent position" and some as "occasional" or "inconstant" or "heard only at times"; a few "disappeared on holding the breath" or on "deep inspiration." In most cases the murmur was located at the apex but occasionally in the third or fourth interspace and once in a while "near the sternal margin." The doctor's diagnosis of the murmur was usually given as "functional" or "insignificant," but in a few cases the qualifying remarks included "mitral regurgitation." The murmur was overlooked in about one-half of the cases on the first examination and was reported only on the second examination which was requested because of a previous record. The previous record was quite variable, sometimes listing as many as six different murmurs, varying from presystolic or diastolic variously located to inconstant pulmonary murmurs. Quite a number of the insured in this class who were given substandard policies subsequently applied for a lower rating and upon medical investigation, such requests were usually granted, indicating that the murmurs were not only inconstant at one examination but also on repeated examinations.

There were only three deaths equivalent to a mortality ratio of 89% among the cases with this murmur on which standard insurance was issued, despite the fact that over 35% of the cases described as having the murmur were issued at

standard rates. In the Medical Impairment Study the mortality experience under standard issues with this murmur showed a mortality ratio of 79% (43 deaths). In his note on "Functional Heart Murmurs" (T.A.S.A., Vol. XXXVIII, p. 141), Dr. A. Hunter gave the experience of four large companies under standard issues with this murmur, covering the issues of 1925–1935 traced to anniversaries in 1936, as 92% (89 deaths).

The Metropolitan's experience on cases rated substandard is given in Table II.

The mortality ratio of 111% for the issues of 1925–1939 while based on 29 deaths only is somewhat lower than the average rating (about +55) assigned to the cases included in this experience. The elimination from this experience of the cases with a history of rheumatism (rated less than +15) reduces the mortality ratio on the issues of 1925–1939 to about 100%.

The Medical Impairment Study experience under substandard issues with this murmur showed a mortality ratio of 137% (59 deaths).

The small but favorable Metropolitan experience under substandard issues when considered in conjunction with the generally favorable experience under standard issues lends considerable support for the belief that this murmur is extracardial and unimportant and that most of the applicants who have it can be accepted for standard insurance. One important exception to this conclusion would be the murmur coded as inconstant because it is brought out only by vigorous exercise, even though it is then heard in all positions, persists for an appreciable time, and may be transmitted. Such a murmur should be regarded with suspicion and, if there is also a history of rheumatism, should unquestionably be underwritten as an important murmur and not coded in this group.

It may be of interest to note that if all of the cases which the Metropolitan accepted at substandard rates had been issued at standard rates, the resulting mortality ratio on standard issues would have been 108% (32 deaths); if all of the

TABLE II

Inconstant Systolic Apex Murmur, without Hypertrophy
Experience on Substandard Issues of 1925–1939 Traced to December 31, 1941

	Mortality Ratio			Number of Policies Terminated by Death		
	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939
Experience by age groups at issue:						
Under 40	241%	*( )%	123%	14	2	16
40 and over	*( )	*( )	99	6	7	13
All ages	156	*( )	111	20	9	29
Experience by policy year duration:						
1–5	*( )	*( )	71	3	7	10
6 and over	206	*( )	158	17	2	19
All ages	156	*( )	111	20	9	29

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

companies which contributed their material to the Medical Impairment Study had likewise issued standard policies in cases which they accepted at substandard rates, the resulting mortality ratio on standard issues would have been only 104% (102 deaths).

The Metropolitan's small substandard experience presented here points to higher relative mortality at ages under 40 at issue than at ages 40 and over at issue and it was in the age group under 40 at issue that all the extra deaths from heart disease occurred. In the Medical Impairment Study experience under both standard and substandard issues the relative mortality at ages 40 and over at issue was higher than at ages under 40 at issue. In the four large companies' experience under standard issues, cited by Dr. Hunter, there was no definite trend in the mortality by age groups at issue. In our opinion the somewhat higher mortality at ages under 40 in the Metropolitan's substandard experience is probably due to the inclusion in the class of some cases with a quiet murmur brought out by exercise—essentially cases with mitral regurgitation.

As regards the causes of death in the Metropolitan's substandard experience on these cases without hypertrophy, there were ten deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 5½ expected deaths from these causes.

There was a very small number of cases with the inconstant systolic apex murmur on which some degree of hypertrophy, mostly very slight, was reported. These cases were all accepted at substandard rates and there was only one death among them.

Constant Systolic Apex Murmur Transmitted to the Left and/or Mitral Regurgitation

Prior to 1935 cases on which this murmur was described as constant systolic apex murmur transmitted to the left, even when qualified by the term mitral regurgitation, were classi-

fied separately from cases on which descriptive details of the murmur were not given and it was simply termed mitral regurgitation. Since 1935, however, these two types of cases have been classified together.

In our standard and substandard Ordinary issues of 1925–1939 cases with constant systolic apex murmur transmitted to the left and/or mitral regurgitation (with and without other impairments) occurred with a frequency of about 210 per 100,000 policies. They accounted for nearly 50% of the accepted heart murmurs. Of the cases so classified but without a history of rheumatism or other infectious disease, over 75% were rated from +55 to +100, and practically all of the remainder were rated from +105 to +150; the average rating for the cases thus classified was about +110. The average rating for the cases which prior to 1935 had been reported simply as mitral regurgitation was also +110. This group included a significant proportion of cases with a history of rheumatism or other infectious disease, the rating for which was less than +15.

In the vast majority of the cases classified in this group, as constant systolic apex murmur transmitted to the left and/or mitral regurgitation without a history of rheumatism or other infectious diseases, the transmission was clearly stated as being to the left or to the axilla, but at times it was also given as slightly upward or to the right or downward. Some of the murmurs were also heard in the back. The murmurs were frequently described as mitral systolic murmurs at the apex or mitral regurgitation and some as mitral regurgitation, slight, moderate, or marked, followed by a fuller description of the murmur. The murmurs were recorded as soft, slight, blowing, very loud, or as entirely replacing first sound. On the whole, this class appears to be reasonably homogeneous, especially as all cases with a history of rheumatism, chorea, tonsillitis or other streptococcic infections were rigidly excluded.

The cases issued prior to 1935 which were classified as

mitral regurgitation were in the great majority reported as "mitral regurgitation" or "mitral insufficiency" without further qualifying remarks. Some of them were described as slight or moderate, some were reported as "mitral regurgitation at the apex," or "mitral regurgitant murmur," or "systolic murmur transmitted." In some cases there was both an anatomical diagnosis and a description of the murmur although not sufficiently detailed to permit classification as constant systolic apex murmur transmitted to the left. In a number of cases, although the diagnosis was mitral regurgitation, the murmur was described as heard at places other than the apex and it would appear that a number of aortic murmurs may have been included in this class. While in the past mitral regurgitation was regarded as a reasonably pure and important pathological entity, this concept is no longer generally accepted. It is now considered that, in rheumatic heart disease, at least, stenosis of the valve fairly early enters the pathological picture, the result of progressive deformity of the valve cusps or of recurrent infection. Having accepted without further study cases diagnosed as mitral regurgitation by older physicians brought up in the earlier concept, we have undoubtedly accepted a goodly proportion of mitral stenoses or mixed lesions in which the systolic murmur was the outstanding and in the doctor's mind the significant finding. Another difference between the cases coded as constant systolic apex murmur transmitted to the left and/or mitral regurgitation and cases arising from the issues of 1934 and prior which were coded simply as mitral regurgitation is that the latter included cases with a history of rheumatism, chorea, tonsillitis, or streptococcic infections, where the rating for the history did not exceed +15.

The Metropolitan's experience on cases rated substandard with the constant systolic apex murmur transmitted to the left and/or mitral regurgitation is given in Table III. This experience does not include cases reported simply as mitral regurgitation, issued prior to 1935. The experience on the latter type of case is shown in Table VI.

It will be noted that the mortality ratios shown in Table III for all ages combined are, even in the category without hypertrophy, considerably higher than the average rating (about +110) assigned to the cases included in this experience.

In the Medical Impairment Study the experience under substandard issues with this murmur without hypertrophy was shown subdivided as between occupations involving light work and heavy work; the former showed a mortality ratio of 218% (1,095 deaths) while the latter showed a mortality ratio of 293% (136 deaths). The mortality ratio for all occupations combined was 224% (1,231 deaths). The Metropolitan's contribution included in the experience for all occupations combined showed a mortality ratio of 248% (63 deaths); the higher mortality ratio on the Metropolitan's material reflected a higher proportion of occupations involving heavy work and a higher proportion of the experience at ages under 40 at issue. The Medical Impairment Study experience on substandard issues with this murmur and hypertrophy showed a mortality ratio of only 234% (394 deaths) for cases with slight hypertrophy, but a mortality ratio of 476% (196 deaths) for cases with moderate hypertrophy.

In his paper on "Heart Murmurs," Dr. A. Hunter gave (T.A.S.A. XXXVII, p. 380) the experience of the New York Life on substandard issues with this murmur; this experience on cases without hypertrophy issued 1915–1924 traced to anniversaries in 1935 showed a mortality ratio of 213% (1,132 deaths), while that on issues of 1925–1934 traced to anniversaries in 1935 showed a mortality ratio of 204% (258 deaths). The corresponding experience on cases with slight hypertrophy showed a mortality ratio of 233% (222 deaths) on the issues of 1915–1924, but a mortality ratio of 327% (116 deaths) on the issues of 1925–1934. On cases with moderate hypertrophy the New York Life's experience on the issues of 1915–1924 gave a mortality ratio of 327% (144 deaths), while on the issues of 1925–1934 the mortality ratio was 466% (53 deaths).

More recently in a note entitled "Mortality under the most

TABLE III

Constant Systolic Apex Murmur Transmitted to the Left and/or Mitral Regurgitation
Without History of Rheumatism or other Infectious Disease
Experience on Substandard Issues of 1925–1939 Traced to December 31, 1941

	Mortality Ratio			Number of Policies Terminated by Death		
	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939
(A) CASES WITHOUT HYPERTROPHY						
Experience by age groups at issue:						
10–29	452%	364%	410%	67	49	116
30–39	368	346	359	51	37	88
40–49	185	194	189	34	28	62
50 and over	115	*( )	118	10	8	18
All ages	291	270	282	162	122	284
Experience by policy year duration:						
1–5	327	275	295	66	92	158
6 and over	270	256	267	96	30	126
All ages	291	270	282	162	122	284

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

TABLE III Continued

	Mortality Ratio			Number of Policies Terminated by Death		
	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939
(B) CASES WITH SLIGHT HYPERTROPHY						
Experience by age groups at issue:						
Under 40	416%	*( )%	360%	22	6	28
40 and over	328	*( )	318	16	9	25
All ages	374	274	339	38	15	53
Experience by policy year duration:						
1–5	637	353	497	24	13	37
6 and over	219	*( )	195	14	2	16
All ages	374	274	339	38	15	53

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

TABLE III Continued

	Mortality Ratio			Number of Policies Terminated by Death		
	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939	Issues of 1925–1931	Issues of 1932–1939	Issues of 1925–1939
(C) CASES WITH MODERATE HYPERTROPHY (Issues of 1925–34 for the most part)						
Experience by age groups at issue:						
Under 40	639%	1010%†	740%†	17	10†	27††
40 and over	*( )	*( )†	455†	9	5†	14††
All ages	511	915†	609†	26	15†	41††
Experience by policy year duration:						
1–5	670	*( )†	724††	13	9†	22††
6 and over	413	*( )†	515††	13	6†	19††
All ages	511	915†	609††	26	15†	41††

†Issues of 1932–1934 for the most part.
††Issues of 1925–1934 for the most part.
*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

common type of heart murmur" (T.A.S.A. XLII, p. 66), Dr. A. Hunter presented the New York Life's experience with this murmur under substandard issues of 1925–1939 carried to anniversaries in 1940. This experience parallels that of the Metropolitan's very closely, as a comparison of the two set forth in Table IV clearly shows.

In cases without hypertrophy, the higher mortality ratio for all ages combined in the Metropolitan's experience reflects a preponderance of the experience at ages under 40 at issue, where the relative mortality is very much higher than at ages 40 and over at issue; likewise, the somewhat higher mortality ratio for ages under 40 at issue in the Metropolitan's experience probably reflects a larger proportion of the experience at ages under 30 at issue, where the relative mortality is higher than at ages 30–39 at issue. The experience of the two companies on cases with slight hypertrophy is also very similar and lends further weight to Dr. A. Hunter's opinion that the relatively low mortality experienced on this class in the Medical Impairment Study (234% with 394 deaths) was probably due to the inclusion of many carefully selected cases where the hypertrophy was either very slight or non-existent. The experience of the two companies further points to the conclusion that the effect of hypertrophy on the mortality of cases with a constant systolic apex murmur transmitted to the left is probably greater than has been supposed. The presence of demonstrable hypertrophy is frequently given by clinicians as one of the criteria for the diagnosis of mitral insufficiency. It is questionable whether any applicant with a heart murmur of presumed mitral insufficiency and also showing hypertrophy is at all acceptable for insurance. This may well be the eventual differentiation between a heart that has been damaged but is still working well and one that has begun to show the ultimately deleterious effect of the underlying pathology.

If we consider accepting applicants with a constant systolic apex murmur transmitted to the left with slight hypertrophy, an additional rating for slight hypertrophy of about +65

TABLE IV

Comparison of the Metropolitan's and New York Life's Experience Under the Constant Systolic Apex Murmur Transmitted to the Left
Without History of Rheumatism or other Infectious Disease

Metropolitan—Issues of 1925–1939 traced to December 31, 1941, except on cases with moderate hypertrophy on which only the issues of 1925–1934 for the most part were studied
New York Life—Issues of 1925–1939 traced to anniversaries in 1940, except on cases with moderate hypertrophy on which only the issues of 1925–1930 were studied

	Mortality Ratio			Number of policies (or amount of insurance) terminated by death		
	Metropolitan	New York Life		Metropolitan	New York Life	
	By number	By number	By amount	Number	Number	Amount ($1000)
(A) CASES WITHOUT HYPERTROPHY						
Experience by age groups at issue:						
10–39	386%	303%	348%	204	171	604
40–49	189	167	266	62	103	817
50 and over	118	152	128	18	91	414
All ages	282	205	228	284	365	1835
Experience by policy year duration:						
1–5	295	215	253	158	177	904
6 and over	267	197	208	126	188	931
All ages	282	205	228	284	365	1835

TABLE IV Continued

	Mortality Ratio			Number of policies (or amount of insurance) terminated by death		
	Metro-politan	New York Life		Metro-politan	New York Life	
	By number	By number	By amount	Number	Number	Amount ($1000)
(B) CASES WITH SLIGHT HYPERTROPHY						
Experience by age groups at issue:						
10–39	360%	394%	408%	28	72	254
40–49	365	310	338	19	57	256
50 and over	*( )	260	355	6	35	206
All ages	339	327	365	53	164	716
Experience by policy year duration:						
1–5	497	410	501	37	90	433
6 and over	195	262	258	16	74	283
All ages	339	327	365	53	164	716

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

TABLE IV Continued

	Mortality Ratio			Number of policies (or amount of insurance) terminated by death		
	Metro-politan	New York Life		Metro-politan	New York Life	
	By number	By number	By amount	Number	Number	Amount ($1000)
(C) CASES WITH MODERATE HYPERTROPHY						
Experience by age groups at issue:						
10–39	740%	554%	493%	27	34	86
40–49	471	341	700	12	23	149
50 and over	*( )	*( )	*( )	2	6	11
All ages	609	358	421	41	63	246
Experience by policy year duration:						
1–5	724	477	540	22	35	138
6 and over	515	273	328	19	28	108
All ages	609	358	421	41	63	246

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

appears to be in order at ages under 40 at issue, while an additional rating of at least +100 is indicated at ages 40 and over at issue. In recent years neither the Metropolitan or the New York Life has accepted applicants with moderate hypertrophy and the experience of the two companies on carefully selected cases insured in the past (1925–1934 in the Metropolitan and 1925–1930 in the New York Life) appears to show that the mortality thereunder is definitely outside the acceptable range.

A comparison of the recent experience of the Metropolitan and the New York Life on cases without hypertrophy with the corresponding experience in the Medical Impairment Study discloses that there has been a remarkable increase in the relative mortality under this impairment at ages under 40 at issue, from the period of years covered by the Medical Impairment Study (1909–1927) to the period covered by the recent Metropolitan and New York Life investigations (1925–1941), as indicated in the table below:

A similar comparison between the average rates of mortality on standard Ordinary risks during the period of years covered by the Medical Impairment Study and the period of years covered by the recent Metropolitan and New York Life investigations explains the increase in the relative mortality at ages under 40 at issue. Such a comparison shows that for a group of standard Ordinary risks at ages under 30 at issue, mortality has improved about 40%; for a similar group at ages 30–39 at issue, mortality has improved about 20%; for similar groups at ages 40–49 and 50–59 at issue mortality has improved 6% and 4% respectively. Thus, while there has actually been little or no change in the absolute mortality rates of persons at ages under 50 with a constant systolic apex murmur transmitted to the left, the level of standard mortality for ages under 40 at issue has decreased about 30% so that the relative mortality of persons with this impairment at ages under 40 at issue has been correspondingly increased. There has been only a slight change in the level of standard mortality at ages 40–49 at issue, so that the relative mortality

TABLE V

Change in the relative mortality by age groups on the constant systolic apex murmur transmitted to the left, without hypertrophy

	Medical Impairment Study Issues of 1909–1927 to anniversaries in 1928	Metropolitan Issues of 1925–1939 to December 31, 1941	New York Life Issues of 1925–1939 to anniversaries in 1940
Age group at issue:			
Under 40	248% (766)	386% (204)	303% (171)
40–49	190 (280)	189 (62)	167 (103)
50 and over	198 (185)	118 (18)	152 (91)

Figures in parentheses indicate number of policies terminated by death.

at these ages has remained about the same. However, there appears to have been an improvement in the absolute mortality rates of persons with this impairment at ages 50 and over at issue. We believe that this improvement in mortality reflects largely the more careful selection procedures adopted for risks at these ages beginning about 1925, as exemplified by the routine requirements at these ages of blood pressure determinations and urinalyses.

In recent years (say, since 1935) the more searching heart examinations have apparently had some favorable effect on the mortality experienced at ages under 40, as may be seen from a comparison of the Metropolitan's experience on the issues of 1925–1931 with that on the issues of 1932–1939 (see Table III). In his note on "Mortality under the most common type of heart murmur," Dr. A. Hunter stated (T.A.S.A. XLII, p. 66) that the New York Life's relatively small experience on policies with this impairment issued in the last few years had also shown a decided tendency towards lower mortality.

However, even after allowance for the more favorable mortality on recent issues due to improved underwriting of heart impairments, the relative mortality under this impairment at ages under 40 at issue is likely to remain quite high, possibly as high as 325% on cases without hypertrophy. This does not necessarily mean that applicants under 40 with a constant systolic apex murmur transmitted to the left without hypertrophy should be rejected, especially if we bear in mind that the absolute mortality rates among such persons have not increased. In the final analysis the answer to the question whether a particular type of risk should be accepted or rejected depends upon the sufficiency of the premiums charged to cover the mortality anticipated on that type of risk. If the premiums charged were sufficient in the past to cover the mortality on the particular type of risk, and if other things being equal there has been no material change in such mortality, it would be reasonable to continue accepting that type of risk, even though such action might involve the taking of

risks with a considerably higher percentage extra mortality at the younger ages than at the older ages. In other words, it may be necessary to abandon the concept of a substandard class as including risks with the same percentage extra mortality at all ages and hence accept at the younger ages risks with a percentage extra mortality that would call for rejection at the older ages. We have, therefore, considered adopting as a criterion of eligibility for each substandard class, the congruence between the mortality anticipated on a particular type of risk and the average mortality actually experienced in each substandard class on policies issued in the same broad age groups. For instance, our highest substandard class was in its original conception intended for risks whose mortality was expected to fall in the range of 205% to 250% of standard; as the level of standard mortality fell and we did not wish to become more strict in our acceptance of substandard risks, the limits of this substandard class were extended to cover the range of 230% to 275% of standard, which meant that the risks assigned thereto under the liberalized limits would tend to have more nearly the same absolute mortality as had previously been acceptable in this class; at the same time we have begun to take into consideration the actual relationship by age groups at issue between the mortality experienced in each substandard class and the corresponding mortality on standard risks, which indicated that the actual mortality in our highest substandard class varied from 275% to 325% of standard at ages under 30 at issue, from 250% to 300% at ages 30–39 at issue, from 225% to 275% at ages 40–49 at issue, and from 175% to 225% at ages 50 and over at issue. If the limits for acceptance in this substandard class were fixed at the upper limit for each age group at issue, we would clearly be in position to offer insurance in our highest substandard class to carefully selected applicants under 40 with a constant systolic apex murmur transmitted to the left, without hypertrophy.

In the Metropolitan's experience on cases with the constant systolic apex murmur transmitted to the left, without hyper-

trophy, there were 176 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 20½ expected. This accounts for about 85% of the extra mortality on these cases. In view of the nature of this extra mortality, it is not surprising that there has been little, if any, change in the absolute mortality rates of persons with this impairment.

The Metropolitan's experience on cases arising out of the substandard issues of 1934 and prior which were reported simply as mitral regurgitation is indicated in Table VI.

TABLE VI

Mitral Regurgitation without Hypertrophy

(including histories of rheumatism or other infectious disease if rated less than +15)

Experience on substandard issues of 1925–34 traced to December 31, 1941

	Mortality Ratio	Number of Policies terminated by death
Experience by age groups at issue:		
Under 40	550%	141
40 and over	335	58
All ages	463	199
Experience by policy year duration:		
1–5	495	88
6 and over	441	111
All ages	463	199

The elimination from the above experience of cases with a history of rheumatism (rated less than +15) does not reduce the mortality ratios materially. Our opinion is that the very high mortality in this experience reflects chiefly the inclusion in this group of persons with mitral stenosis, and even some aortic murmurs.

In this experience on cases reported as mitral regurgitation, there were 139 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 8 expected. This accounts for about 85% of the extra mortality on these cases.

Constant Systolic Apex Murmur and/or Mitral Regurgitation with History of Rheumatism, Chorea, Tonsillitis or Other Streptococcic Infection

In our standard and substandard Ordinary issues of 1925–1939 cases with constant systolic apex murmur and/or mitral regurgitation with a history of rheumatism, chorea, tonsillitis or other streptococcic infection occurred with a frequency of about 35 per 100,000 policies, accounting for about 8% of the accepted heart murmur. About 62% of the cases with this impairment were rated from +55 to +100 and the remainder from +105 to +150. The average rating was about +120.

The vast majority of the murmurs in this class were described as systolic at the apex but a few were also reported as "mitral systolic murmurs." Transmission was usually towards the left or to the axilla and occasionally to the back. The murmur was generally found on the first examination, perhaps because of the history of rheumatism.

In about 70% of the cases, the history was given as "rheumatism" which was sometimes also described as inflammatory rheumatism, acute rheumatic fever or acute arthritis. In about 25% of the cases with a history of rheumatism, there was a record of tonsillectomy. The histories other than rheumatism included a number of cases of scarlet fever, diphtheria, frequent sore throat, tonsillitis and tonsillectomy. Chorea was very rarely admitted. In most cases the history was readily admitted and in some the applicant acknowledged that he had been aware of a valvular lesion prior to examination.

The Metropolitan's experience on cases rated substandard with the constant systolic apex murmur and/or mitral regurgitation with a history of rheumatism, chorea, tonsillitis and other streptococcic infection is given in Table VII.

TABLE VII

Constant Systolic Apex Murmur and/or Mitral Regurgitation with a History of Rheumatism, Chorea, Tonsillitis or Other Streptococcic Infection
Experience on Substandard Issues of 1925–1939 Traced to December 31, 1941

	Mortality Ratio			Number of Policies Terminated by Death		
	Ages under 40	Age 40 and over	All ages	Ages under 40	Age 40 and over	All ages
(A) CASES WITHOUT HYPERTROPHY						
With history of rheumatism without tonsillectomy	517%	358%	467%	35	11	46
With history of rheumatism and tonsillectomy			400			11
With history of rheumatism with and without tonsillectomy	480	385	453	43	14	57
With history of infectious disease other than rheumatism			294			15
With history of rheumatism or other infectious disease	444	314	407	56	16	72

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

TABLE VII Continued

	Mortality Ratio			Number of Policies Terminated by Death		
	Ages under 40	Age 40 and over	All ages	Ages under 40	Age 40 and over	All ages
(B) CASES WITH HYPERTROPHY						
With history of rheumatism or other infectious disease and slight hypertrophy			323%			17
With history of rheumatism or other infectious disease and moderate hypertrophy			765			13
With history of rheumatism or infectious disease and severe hypertrophy			1429			12
With history of rheumatism or infectious disease and any hypertrophy	759%	*( )	538	40	3	43
With history of rheumatism and any hypertrophy	796	*( )	563	34	3	37

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

It will be noted that the mortality ratios shown in Table VII for cases with a history of rheumatism are very much higher than for cases with a history of other infectious

TABLE VIII

Comparisons of Metropolitan and Medical Impairment Study Experiences on Constant Systolic Apex Murmur and/or Mitral Regurgitation with a History of Rheumatism, Chorea, Tonsillitis, or other Streptococcic Infection

Metropolitan—Issues of 1925–1939 traced to December 31, 1941
Medical Impairment Study—Issues of 1909–1927 traced to anniversaries in 1928

	Mortality Ratio		Number of Policies terminated by death	
	Met.	M. I. S.	Met.	M. I. S.
(A) CASES WITHOUT HYPERTROPHY				
With a history of rheumatism without tonsillectomy	467%	332%	46	287
With a history of rheumatism and tonsillectomy	400	325	11	20
With a history of rheumatism with and without tonsillectomy	453	331	57	307
With a history of infectious disease other than rheumatism	294	189	15	103
With a history of rheumatism or other infectious disease	407	278	72	410
(B) CASES WITH HYPERTROPHY				
With a history of rheumatism and slight or moderate hypertrophy	451	422	26	158
With a history of rheumatism or other infectious disease and slight or moderate hypertrophy	431	337	30	192

disease and that the mortality on cases with hypertrophy is very high. Since a much larger experience under the constant systolic apex murmur and/or mitral regurgitation with a history of rheumatism, chorea, tonsillitis, or other streptococcic infection was given in the Medical Impairment Study in corresponding subdivisions, a comparison of the two experiences set forth in Table VIII is instructive.

Insofar as the figures in the relatively small but more recent Metropolitan experience go, they tend to confirm the findings in the Medical Impairment Study. Judged by the Metropolitan's experience on the issues of 1925–39 with and without a history of rheumatism (see Tables IV and VII), the effect of such a history on the mortality under the constant systolic apex murmur and/or mitral regurgitation is, if anything, greater than indicated in the Medical Impairment Study; this study pointed to an additional rating of about +100 for a history of rheumatism and the Metropolitan's small experience on the issues of 1932–1939 with and without a history of rheumatism indicates that such an additional rating is undoubtedly necessary. What appears even more definite is that presumed rheumatic heart disease, that is, a systolic apex murmur with a definite history of acute rheumatic fever is uninsurable at ages under 40, even if there is no hypertrophy. Neither the Metropolitan's experience nor the larger data in the Medical Impairment Study give any clear-cut indication as to the relative effect of a history of rheumatism on mortality at different ages at issue. There is likewise no indication either in the Metropolitan or the Medical Impairment Study that a history of tonsillectomy has any effect on the mortality of cases with a history of rheumatism.

The effect of infectious diseases other than rheumatism on the mortality under the constant systolic apex murmur and/or mitral regurgitation appears to be quite small, if not negligible.

The effect of hypertrophy, as stated in the Medical Impairment Study, is apparently worse on cases with a history of rheumatism than on cases without any history of infectious

disease. Some indication of this can be gained from the Metropolitan's experience with and without hypertrophy on cases with and without a history of rheumatism as well as from the New York Life's experience with the constant systolic apex murmur and a history of rheumatism or other infectious disease, cited by Dr. A. Hunter in his paper on "Heart Murmurs" (T.A.S.A. XXXVII, p. 380). This latter experience on cases without hypertrophy issued 1915–1924 traced to anniversaries in 1935 showed a mortality ratio of 213% (235 deaths), while that on the issues of 1925–1934 traced to anniversaries in 1935 showed a mortality ratio of 205% (54 deaths). The corresponding experience on cases with slight hypertrophy showed a mortality ratio of 272% (107 deaths) on the issues of 1915–1924, but a mortality ratio of 394% (38 deaths) on the issues of 1925–1934. On cases with moderate hypertrophy the New York Life's experience on the issues of 1915–1924 gave a mortality ratio of 410% (117 deaths) while on the issues of 1925–1934 the mortality ratio was 582% (37 deaths).

In the Metropolitan's experience on cases with the constant systolic apex murmur and/or mitral regurgitation with a history of rheumatism but without hypertrophy there were 39 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 2½ expected. This accounts for nearly 85% of the extra mortality on cases with this heart murmur and a history of rheumatism.

Systolic Apex and Other Heart Murmurs on Which Details Were Not Obtained

In our standard and substandard Ordinary issues of 1925–1939 cases with a systolic apex murmur on which other details were not obtained (Class 5) occurred with a frequency of about 70 per 100,000 policies, while murmurs on which sufficient information was not obtained to classify them with any more carefully defined group (Class 6) occurred with a frequency of about 60 per 100,000 policies. Systolic apex and other heart murmurs on which details were not obtained to-

gether accounted for about 29% of the accepted heart murmurs. This situation reflects largely the fact that practical considerations of the cost of another medical examination and of the unwillingness of the applicant to be re-examined frequently precluded the obtaining of more accurate data, especially in cases where an underwriting decision could be reached without additional information.

Of the cases included in Class 5, i.e., systolic apex murmur without sufficient detail to permit inclusion in Classes 1, 2 or 3, about 6% were rated standard, 9% were rated from +30 to +50, 68% were rated +55 to +100, and 17% were rated from +105 to +150. The average rating for the cases rated +50 or less was +25, while for the cases rated +55 to +150 it was +105. Included among the cases rated +105 to +150, was a number with a history of rheumatism or other infectious disease, where the rating for the history did not exceed +15. Of the cases included in Class 6, i.e., other heart murmur without details, about 24% were rated standard, 26% were rated +30 to +50, 41% were rated from +55 to +100, and 9% were rated from +105 to +150. The average rating for the cases rated +50 or less was +20, while for the cases rated from +55 to +150 it was +105. Included among the cases rated +105 to +150, was a number with a history of rheumatism or other infectious disease, where the rating for the history did not exceed +15.

The cases in Class 5 comprised a conglomeration of variously reported heart lesions. In most cases the examining physician omitted stating whether or not the systolic apex murmur was constant or inconstant, and whether it was transmitted to the axilla. Frequently, the direction of transmission was given as "upwards," "towards the base," "downward," "towards the sternum," "towards the aortic area," or "to the right." A number of murmurs included in this class were reported as definitely inconstant or heard only on exercise or elicited by exercise, but were further described as transmitted, generally not towards the axilla. Judging from the descriptions such as "murmur not heard at full inspiration

or expiration," some cardio-respiratory murmurs must have been included in this class. The actual descriptions of the murmur varied widely. Many of the cases in this class were obviously "formulae" reports; for instance, a considerable number were from Chicago where we know that many of our doctors routinely reported presumed "mitral regurgitation" as a systolic murmur transmitted towards the base. That these cases represent stereotyped reports is also brought out by the fact that some of the physicians whose cases were included in this class had few, if any, cases reported in Class 1 or 3 and vice versa. In a number of instances, the murmur was reported only on the second examination called for by a previous record, and not infrequently the report on which our decision was based was more favorable than the record. It is clear, on review, that this class really included a large proportion of constant systolic apex murmurs, presumably transmitted to the left, a number of constant systolic apex murmurs not transmitted, and inconstant systolic apex murmurs as well as some murmurs of a cardio-respiratory nature, all reported in terms which did not fit the definitions for Classes 1, 2 or 3.

Class 6 included, for the most part, cases on which the details given were either not sufficient or contradictory, so that they could not be classified in categories 1, 2, 3 or 5. There were relatively few cases which were literally "without details" and these were chiefly murmurs described as in the 2nd, 3rd, or 4th interspace or along the sternum or beneath or above the sternum or below the ensiform cartilage, the timing not being given. Most of them were probably pulmonic murmurs. Some were definitely called tricuspid or congenital or pulmonic. Pulmonic murmurs, if inconstant, were apparently routinely included in this class until 1935. Many of the murmurs were systolic in time and were described as "slight" or "soft" or "loud" or "blowing" but a few were given as "impure" or "rubbing first sound." Most of the murmurs on which details were given were probably constant systolic apex murmurs transmitted to the left where the doctor did not state that the murmur was systolic or at the apex, but other infor-

mation nevertheless led the underwriter to appraise the case as such. The cases in this class illustrate very well the difficulties of obtaining reports on heart murmurs which would be sufficiently precise and couched in such uniform terms as would permit a lay person to code them into groups reasonably homogeneous from the clinical or underwriting viewpoint.

The experience on these two classes of murmurs is, therefore, of little, if any, value for judging the mortality of definite clinical entities. The principal interest which we had in this experience was to see whether the underwriters had been successful in establishing the probable significance of the murmurs without recourse to further inquiries or second examinations. We may assume that where the underwriter rated cases standard or from +30 to +50, he reached a decision that the murmur was unimportant; where he rated cases from +55 to +100 or from +105 to +150 he must have regarded the murmur as serious. A study of the experience according to the rating assigned gives some indication of the underwriter's ability to differentiate between important and unimportant murmurs.

The Metropolitan's experience on cases with the systolic apex murmur and other heart murmurs without hypertrophy on which details were not obtained is given in the table below separately for each class and separately for cases rated +50 or less and for those rated +55 to +150.

It will be noted that the mortality ratios shown in Table IX for cases rated +50 or less correspond closely to the average ratings (+20 or +25) assigned to the cases in this group, but that the mortality ratios shown for cases rated +55 to +150 are considerably higher than the average ratings (+105) assigned to the cases included in this latter group. The elimination of the cases with a history of rheumatism (rated less than +15) reduces the mortality ratio on the cases in Class 5, which were rated +55 to +150 from 231% to 225%. On cases in Class 6, which were rated +55 to +150 the mortality ratio is similarly reduced from 309% to 300%; on all the

TABLE IX

Systolic Apex Murmur and other Heart Murmurs on which Details were not obtained, without Hypertrophy

Experience on Standard and Substandard Issues of 1925–1939 Traced to December 31, 1941

	Mortality Ratio			Number of policies terminated by death		
	Ages under 40	Age 40 and over	All ages	Ages under 40	Age 40 and over	All ages
(A) Systolic Apex Murmur without Details (Class 5)						
Cases rated +50 or less	*( )	*( )	*( )	5	3	8
Cases rated +55 to +150	241	217	231	69	45	114
(B) Other Heart Murmur without Details (Class 6)						
Cases rated +50 or less	160	*( )	127	15	6	21
Cases rated +55 to +150	346	258	309	54	29	83
(C) Systolic and other Heart Murmur without Details (Classes 5 and 6 combined)						
Cases rated +50 or less:						
Issues of 1925–1931	*( )	*( )	127	8	6	14
Issues of 1932–1939	189	*( )	120	12	3	15
Issues of 1925–1939	159	*( )	123	20	9	29
Cases rated +55 to +150:						
Issues of 1925–1931	301	226	268	93	53	146
Issues of 1932–1939	224	247	233	30	21	51
Issues of 1925–1939	278	232	258	123	74	197

*( ) indicates that the mortality ratio would have been based on less than 10 deaths.

cases in Classes 5 and 6 which were rated +55 to +150, the mortality ratio is reduced from 258% to 250%, by eliminating the cases with a history of rheumatism.

That the mortality ratio of 278% at ages under 40 at issue in Table IX for the group of cases rated +55 to +150 is more favorable than the corresponding ratio for the constant systolic apex murmur transmitted to the left (see Table III) is probably due to the inclusion, in the former group (particularly in Class 6) of many cases with pulmonary and other unimportant murmurs. On the whole, taking into consideration that our medical underwriters were given as a guide ratings for the constant systolic apex murmur transmitted to the left which proved insufficient, it would appear that their accomplishment in distinguishing between the important and unimportant heart murmurs, without calling for additional information on cases where details were not given on the first examination, deserves high commendation.

On cases with the apex systolic murmur or other heart murmur on which details were not obtained (Classes 5 and 6) with slight hypertrophy, nearly all of which were rated +80 or more, the mortality ratio was 333% (25 deaths) as compared with a mortality ratio of 339% (53 deaths) on cases with the constant systolic apex murmur transmitted to the left with slight hypertrophy. On cases in Classes 5 and 6 with moderate hypertrophy, nearly all of which were rated +80 or more, the mortality ratio was 368% (15 deaths) as compared with a mortality ratio of 609% (41 deaths) on cases with the constant systolic apex murmur transmitted to the left with moderate hypertrophy. It is thus clear that the cases of heart murmurs on which details were not obtained with slight or moderate hypertrophy were treated by our medical underwriters as presumed mitral regurgitation and that much the same mortality experience was obtained as in Class 3.

As regards the causes of death on cases in Class 5, without hypertrophy, there were 57 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 11 deaths ex-

pected. This accounts for about 70% of the extra mortality on cases in this class.

On cases in Class 6, without hypertrophy, there were 56 deaths from heart disease (Joint Committee codes 42, 43, 44 and 45) as against 8½ deaths expected from these causes. This accounts for over 75% of the extra mortality on cases in this class.

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The heart murmurs coded in Classes 1, 2, 3, 4, 5 and 6, accounted for over 95% of all of the heart murmurs accepted by our Company during the years 1925–1939. Of the heart murmurs not included in Classes 1, 2, 3, 4, 5 and 6, the commonest one was the pulmonic systolic murmur not transmitted, which occurred with a frequency of about 7 per 100,000 policies in our standard and substandard issues of 1925–1939. Apparently a great many of the pulmonic systolic murmurs were included in the classification of "heart murmur without details." There were only four deaths on the cases with the pulmonic systolic murmur not transmitted which were accepted over this period, and these were all on the substandard issues. There was also one death among the cases with the constant systolic aortic murmur not transmitted.

Summary

The Material

We have set down here the results of an investigation into the mortality of white insured lives presenting on examination some type of systolic murmur over the left side of the chest. The investigation covered policies issued from 1925 through 1939, traced through December, 1941. It was based on our medico-analytical card records coded in accordance with the impairment classifications adopted by the Joint Committee on Mortality in 1925 and revised in 1935. These classifications depend largely upon the description of the murmur as reported by the examining physician. In the course of the

years covered by the investigation our selection and scrutiny of individual cases became progressively more careful.

Attention is called to the fact that the material contained in the classifications studied is quite heterogeneous and that each classification is made up of varying proportions of important and unimportant murmurs. In relation to our underwriting standards, the cases rated +50 or less may be considered as representing the unimportant murmurs and those rated +55 to +150 the important murmurs.

The systolic murmur that is probably of greatest interest to life insurance companies is the constant apex murmur which is transmitted. It is believed to represent some degree of mitral incompetence or mitral regurgitation. Whether through local custom or differences of training in physical diagnosis or acquired habit pattern, examining physicians report such murmurs in widely varying terms. It is possible that some physicians first decide on a diagnosis and then report the murmur which in their minds represents the diagnosis. Whatever the cause of the varying terminology, murmurs which the physician apparently considered to be mitral regurgitation without stenosis were reported to us as "mitral regurgitation," "systolic murmur at apex transmitted to the left," "systolic murmur at apex, transmitted to base," "mitral lesion at apex," "mitral murmur at apex brought out by exercise transmitted to the left," or even "mitral murmur at apex not transmitted—mitral regurgitation." To the doctor and the underwriter these reports all represent the insurance concept of mitral regurgitation, but without further study they would be assigned to different classifications, according to present code definitions.

For this reason, cases classified as "constant, systolic, apex murmur, not transmitted" (Class 1) unquestionably include a definite proportion of murmurs which the examining physician must have considered as representing mitral regurgitation as well as an indeterminate number of cases which probably were mitral regurgitation but were not classified as such because the examining physician did not hear a very low

pitched murmur elsewhere than at the apex. Even in those instances where a history of rheumatism was admitted, pointing to the probability that the murmur was an important one, its transmission was in some cases not evident to the examining physician.

The cases classified as "inconstant, systolic, apex murmur" (Class 2) also include a small proportion of presumably mitral regurgitant murmurs, e.g., those in which the murmur appears only on exercise, though it may then tend to persist and may even be transmitted.

Cases classified as "constant, systolic, apex murmur, transmitted to the left and/or mitral regurgitation" (Class 3) represent on the whole a reasonably homogeneous group but did include at least in the past a small number of stenotic lesions. Prior to 1935, heart lesions reported by the examing physician simply as "mitral regurgitation" were not included in Class 3 but were classified separately. Judging from the results on our material so separately classified, cases reported simply as mitral regurgitation should be kept apart and for insurance purposes no such diagnosis should be accepted without further study. Older doctors brought up on the concept that mitral regurgitation is a definite anatomical lesion may report as such all constant, systolic, apical murmurs, whether or not a diastolic murmur may also be present.

Cases classified as "constant, systolic, apex murmur and/or mitral regurgitation with a history of rheumatism or other infectious disease" (Class 4) represent in about 70% of the cases mitral valve disease of rheumatic origin and some stenotic lesions were also undoubtedly included.

Cases classified as systolic, apex murmur or other heart murmur on which details were not obtained (Classes 5 and 6) are conglomerations of murmurs inadequately reported or incompletely investigated from the viewpoint of the code definitions. A large proportion of the murmurs in these classes were those of presumed mitral regurgitation but a considerable proportion of unimportant murmurs were also included.

The Results

Cases coded as "constant, systolic, apex murmur, not transmitted" (Class 1) without hypertrophy, gave an experience on substandard issues of 1925 to 1939, after elimination of cases with a history of rheumatism, equivalent to about 185% of the expected. In the small experience on the substandard issues of 1932 to 1939 covering the period of more careful heart investigations, the mortality ratio was only about 126%. From this experience and that of other companies, we conclude that if all applicants presenting this murmur were very carefully investigated, they could probably be divided into two groups: (1) those in which the murmur is inconstant, not actually heard at all times and in all positions and, (2) those in which the murmur is constant and after vigorous exercise definitely transmitted. As a result of our experience, we shall consider making more careful examinations looking toward this segregation in order to give standard insurance to applicants in the former group. Cases in the latter group with hypertrophy or a history of acute rheumatic fever would, for rating purposes, be considered as rheumatic mitral disease.

As regards the cases coded as "inconstant, systolic, apex murmur" (Class 2) without hypertrophy, our very small experience on standard issues showed a mortality ratio of 89% based on only three deaths, while the larger experience on substandard issues, after elimination of cases with a history of rheumatism, showed a mortality ratio of about 100%. From this latter experience and that of other companies, we conclude that this murmur can safely be accepted at standard rates. Cases with hypertrophy or a history of acute rheumatic fever would, for rating purposes, be considered as rheumatic mitral disease.

As regards the cases coded as "constant, systolic, apex murmur, transmitted to the left and/or mitral regurgitation" (Class 3) without hypertrophy and without any history of rheumatism or other infectious disease, our experience on substandard issues of 1925 to 1939 was 282% of the expected,

based on 284 deaths; on the more recent substandard issues of 1932 to 1939, covering the period of more careful heart examinations, the experience was 270% of the expected, based on 122 deaths. At ages under 40 at issue, the mortality ratio in the latter group was 356%, based on 86 deaths; at ages 40 to 49 at issue, it was 194%, based on 28 deaths, and at ages 50 and over at issue, it was 122% based on 8 deaths only. From this experience and that of the New York Life recently published by Dr. A. Hunter (T.A.S.A. XLII, p. 66), it is clear that this murmur at ages under 50 at issue represents a serious condition, irrespective of whether it is due to a mitral valve lesion caused by a previous attack of rheumatism of which the applicant may not have been aware or whether it represents a relative incompetence due to ventricular dilatation or a valve deformed or scarred by sclerosis or calcification. The lower mortality of applicants with this murmur at ages 50 and over at issue may indicate that most of the murmurs at these ages were caused by sclerosis of the valve and as such, merely represent some acceleration of the aging process; it is also in part due to the elimination of the worst risks through the routine requirement at these ages of a blood pressure determination and a urinalysis. The very high mortality ratio for ages under 40 at issue in the Metropolitan's experience reflects the fact that standard mortality at these ages has declined about 30% over the years covered by the investigation. Actually, the mortality rates per 1,000 among applicants at ages under 40 at issue with this murmur have remained about the same but their relative mortality has increased because of the decline in the level of standard mortality. The acceptance of these applicants at ages under 40 at issue involves the taking of risks with a higher percentage extra mortality at the younger ages than would generally be considered prudent at the older ages. However, if this practice were adopted, then we could give insurance in our highest substandard class to those applicants under 40 at issue who were engaged in an occupation involving no manual labor, if there was no hypertrophy, if the history was entirely nega-

tive and if there were no other ratable impairments. At ages 40 to 49 at issue, a rating of +100 is indicated while at ages 50 and over at issue, a rating of +50 would appear sufficient.

In those cases in Class 3 which also presented hypertrophy, our experience showed a mortality ratio of 339%, based on 53 deaths where the hypertrophy was reported as slight, and a mortality ratio of 609%, based on 41 deaths, where the hypertrophy was reported as moderate. From this and the experience of other companies, we conclude that even slight hypertrophy should be evidence for the underwriter that the heart is currently showing the effect of damage sustained in the past. We do not feel that applicants under age 50 at issue with presumed mitral regurgitation and even slight hypertrophy are acceptable risks, assuming that an extra mortality of at least 200% calls for rejection at these ages. In the case of applicants at ages 50 and over at issue with presumed mitral regurgitation and slight hypertrophy, an additional rating of +100 for the slight hypertrophy is indicated.

In the class which prior to 1935 included cases reported simply as "mitral regurgitation," our experience on cases without hypertrophy showed a mortality ratio of about 450% after eliminating the cases with a history of rheumatism. This very high mortality, for the most part on cases issued in the days of less intensive heart examinations, is a warning that cases reported as "mitral regurgitation" should not be accepted without further study.

In the class of cases coded as "constant, systolic, apex murmur, and/or mitral regurgitation with a history of rheumatism or other infectious disease" (Class 4), without hypertrophy, our substandard experience on cases with a history of rheumatism alone showed a mortality ratio of 453%, based on 57 deaths; the small experience on cases with a history of infectious disease other than rheumatism showed a mortality ratio of only 294%, based on 15 deaths, or about the same as in Class 3 without hypertrophy. From our experience and the much larger experience in the Medical Impairment Study,

we conclude that an additional rating of +100 for a history of rheumatism is indicated. This means that applicants at ages under 50 at issue with presumed rheumatic mitral disease are not acceptable risks, while selected applicants at ages 50 and over might perhaps be taken at a rating not less than +150. "Selection" in this case probably means identifying those cases in which the rheumatism reported was not acute rheumatic fever but some chronic or subacute arthritis. For histories of infectious disease other than rheumatism, a small rating, say +15, appears appropriate more as a matter of conservatism than from considerations of the mortality experience.

In the classes of cases coded as systolic, apex murmur, without further details or other heart murmur without details (Classes 5 and 6) without hypertrophy, the combined experience on the cases rated +50 or less was 123% of the expected, based on 29 deaths, while the combined experience on the cases rated +55 to +150 was, after elimination of cases with a history of rheumatism, about 250% of the expected, based on 172 deaths. This experience on a very heterogeneous group of murmurs indicates that it has been possible to distinguish between the important and the unimportant heart murmurs on the basis of the information generally available to the underwriter.

Conclusions

1—Clinical evidence and mortality studies have established systolic murmurs in the pulmonic area as clearly unimportant and such indirect data as we have on this point support this conclusion. The experience here presented also adds to the existing evidence that a definitely inconstant systolic apex murmur is unimportant and can be accepted at standard rates. Our experience further points to the conclusion that the murmurs reported as constant, systolic at the apex, not transmitted are, for the most part, unimportant and that if we could, through careful instructions to our examiners or repeated examinations, eliminate the murmurs which are in point of

fact transmitted, the remainder could, in our opinion, be safely accepted at standard rates.

At the other end of the scale are presystolic and diastolic murmurs, constant, systolic murmurs to the right of the sternum, and systolic apex murmurs accompanied by moderate hypertrophy, arrhythmia or poor exercise tolerance. These conditions are considered as not insurable.

Our experience definitely indicates that where the applicant is under age 50, we should now add to this group of murmurs which are not acceptable for insurance any constant, systolic, apex murmur which is transmitted, if there is also evidence of any hypertrophy or a history of acute rheumatic fever at any time. While transmission is usually described as to the left and that direction is considered typical of mitral incompetence, we believe that any transmission in any direction so long as the point of maximal intensity of the murmur is at or near the apex is significant, particularly when our reports are based on examinations by physicians who are not heart specialists.

In this connection we should like here to suggest that any apical, systolic murmur whether described as constant or inconstant with a history of acute rheumatic fever be considered and underwritten as presumed rheumatic mitral disease corresponding to Class 4 of this study.

This leaves for consideration the constant systolic apex murmur which is transmitted and/or mitral regurgitation without hypertrophy and without any history of rheumatism. Our experience as well as that of the New York Life recently published by Dr. A. Hunter (T.A.S.A., XLII, p. 66) indicates a probable percentage extra mortality of about 225% at ages under 40 at issue, about 100% at ages 40–49 at issue, and about 50% at ages 50 and over at issue. It is therefore obvious that at ages under 40 at issue any rateable or other significant condition, if found in conjunction with this murmur, should lead to rejection. At ages 50 and over at issue, cases with slight hypertrophy or with a history of rheumatism (but not both) can probably be accepted at a rating of not less

than +150, since each of these conditions appears to call for an additional rating of about +100 at ages 40 and over.

A history of an infectious disease other than rheumatism does not, on the basis of our experience or that of the Medical Impairment Study, appear to be particularly significant, but nevertheless we feel that in a small number of cases it might be so and hence have suggested a small rating of +15. The principal effect of such a rating will be to render ineligible for standard insurance, cases with an inconstant or a non-transmitted systolic, apex murmur, on which there is also a history of some infectious disease other than rheumatism and this we feel is in keeping with the careful selection of such cases for standard insurance.

A tentative rating schedule for heart murmurs based on the foregoing considerations is given in Appendix I.

2—In order to distinguish between the important and unimportant murmurs which examiners call to our attention, it appears necessary in many cases to make more careful heart investigations than we have made in the past. This is not a very serious problem because the frequency of the cases requiring more careful investigation should not exceed 350 per 100,000 applications received.

The need for more careful examinations arises essentially from the fact that most rateable heart murmurs fall into the category of "borderline" risks; the unimportant heart murmurs are frequently borderline for standard insurance, while the constant, systolic, apex murmur transmitted is, at ages under 40, definitely on the borderline for rejection. We feel that the additional cost of more careful examinations is, therefore, justified not only from the Company's viewpoint as being necessary to determine the proper action on questionable risks, but also as a matter of public policy to avoid "stigmatizing" applicants by either refusing to issue the insurance at standard rates or by outright rejection, on the basis of reported findings which on later study could not perhaps be substantiated. We should also bear in mind that some of our examiners find or

report relatively a very much higher proportion of heart murmurs than others, so that such findings or reports may have different significance depending upon the examining physician.

Dealing first with heart murmurs which may be borderline for standard insurance, we feel that in all questionable cases it will probably be necessary to obtain a heart chart. We would consider any case as questionable where the report of the murmur is not absolutely clear-cut or where there is a history of infectious disease or any other significant impairment. When the murmur is reported as a constant, apex, systolic murmur, not transmitted, it would be advisable to establish that there is actually no transmission, just as when the murmur is reported as inconstant, it may be advisable to establish that it really is inconstant in all positions and after vigorous exercise. We do not feel that a recheck will generally be necessary in the case of systolic murmurs reported in the pulmonic area or in the 2nd or 3rd left interspaces, unless they are also described as being transmitted. The recheck is probably best made through the medium of a heart chart so devised as to bring forcibly to the examining physician's attention all of the points of differentiation which are significant to the underwriter.

In the case of murmurs which are borderline for rejection, we feel that it would be advisable to obtain a heart chart in all cases, especially in order that any diastolic murmur, any evidence of hypertrophy or any history of acute rheumatic fever be brought to light. To identify the murmur in terms of our own categories from the multiplicity of terms used by our physicians, it may either be necessary to ask leading questions on the heart chart or actually to set down our own categories and ask the examining physician to indicate the one into which his report would most nearly fall. In other words, if we are to act on stereotyped reports, we should mould the patterns into which they will fall.

A tentative form of heart chart, such as we have in mind, is given in Appendix II.

3—In order to facilitate the interpretation of future mortality studies of heart murmurs, it would appear desirable to redefine present codes so that each would include more closely related heart conditions and would correspond more nearly to definite underwriting classifications.

As a result of the diverse terminology with which heart murmurs are described to us, the present classifications of heart murmurs have in practice come to include heart conditions of widely varying significance, calling for altogether different ratings. Moreover, different company attitudes toward indefinite reports, reports of heart murmurs where there is a contradictory history, etc., have given rise to different coding practices as between companies. This is illustrated by the Metropolitan's very low frequency of cases coded as pulmonic, systolic murmur, not transmitted (about 7 per 100,000). In other companies this type of murmur is apparently reported and coded more frequently, but in the Metropolitan a large proportion of these murmurs have been described as in the 2nd or 3rd left interspace, without mention of the timing, constancy, or transmission, and consequently they have been included in the classification of "heart murmurs without details." As a result, the frequency of cases included in the latter group has been relatively high in our Company. Another illustration of the differences in coding practices is offered by the low frequency in some companies of cases coded as inconstant systolic apex murmur.

While each application showing a heart murmur is generally coded separately for mortality studies on the basis of the examining physician's report, it will always have been carefully appraised by an underwriter on the basis of more comprehensive considerations. It is our opinion that if such more comprehensive considerations were also kept in mind in defining classifications for mortality studies, we would obtain more homogeneous and significant groupings than at present. While we are not prepared to make any specific recommendations for changing the present procedure for classifying heart murmurs, we believe that a new coding

scheme should comprise fewer classifications, grouping together related or equivalent lesions based on clinical and underwriting estimates of their significance.

* * * * * * *

No paper dealing with the mortality under heart murmurs would be complete without an acknowledgement of indebtedness to Dr. A. Hunter for his outstanding contributions on this subject. In this connection, we also wish to express our appreciation of the courtesy of the New York Life Insurance Company in making available to us certain details of its experience under heart murmurs.

APPENDIX I

PROPOSED RATINGS AND INSTRUCTIONS FOR THE INVESTIGATION OF HEART MURMURS

A. The following ratings apply only to the murmurs described as below where there is no indication of any hypertrophy and no record of more serious heart murmurs or history of rheumatism or other infectious disease. Where there is any hypertrophy or a record of a history of rheumatism, rate as mitral regurgitation. Where there is a record of more serious heart murmur, obtain a second examination. Where there is a history of an infectious disease other than rheumatism (frequent sore throats, scarlet fever, chorea, etc.) add +15 to the rating.

Type of Murmur	Rating	Investigation
Systolic Murmur in Pulmonic Area or in 2nd or 3rd Left Interspace Not Transmitted	+10	Heart chart if described as transmitted
Apex Systolic Murmur, Inconstant	+10	Heart chart
Apex Systolic Murmur, Constant, not transmitted	+25	Heart chart

B. The following ratings apply only to the murmurs described as below where there is no indication of any hypertrophy and no record of more serious heart murmurs or history of rheumatism or other infectious disease. Where there is slight hypertrophy* add +100 to the rating. Where there is a record of more serious heart murmur, obtain a second examination. Where there is a history of rheumatism, add +100 to the rating. Where there is a history of an infectious disease other than rheumatism (frequent sore throats, scarlet fever, chorea, etc.), add +15 to the rating.

Type of Murmur	Rating	Investigation
Apex Systolic Murmur, Constant, transmitted Mitral Regurgitation		Heart chart if no history of rheumatism or infectious disease is noted or details of the murmur are insufficient for accurate classification
In occupations requiring light work only:		
Ages under 40	+225	
Ages 40–49	+100	
Ages 50 and over	+ 50	
In occupations requiring manual labor or other heavy work, even though occupation is not ratable:		
Ages under 40	Decline	
Ages 40–49	+150	
Ages 50 and over	+100	

* Slight hypertrophy — apex not beyond the midclavicular line and not below the 5th interspace; no enlargement (hypertrophy) of the right side of the heart.

C. The following murmurs should be investigated further to establish their nature more definitely:

Basal Murmur in 4th interspace at border of sternum

Aortic Systolic Murmur Inconstant

Inconstant Systolic Murmur heard in 2nd or 3rd right interspace

"Functional" Murmur

Any Murmur reported as a diagnosis without a description of the Murmur, as "Mitral Regurgitation"

D. The following murmurs should be rejected:

Presystolic or Diastolic Murmurs
Any Constant Systolic Murmur to the right of the sternum
Any murmur with moderate or severe hypertrophy
Any murmur with arrhythmia or poor exercise tolerance.

APPENDIX II

PROPOSED HEART CHART

We believe that the points covered below, in the proposed heart chart, will be sufficient to bring out all the significant data. The arrangement, of course, is subject to limitations of space on the form. The diagram should be sufficiently large so that the examiner's location of the apex and the murmur can be accurately indicated. In addition to showing the mid-clavicular line the diagram should also show the location of the mitral, pulmonic and aortic valve areas.

Special Heart Examination

NOTE: For this examination the applicant should be erect either sitting or standing with the chest bared. Every effort must be made to avoid offending the applicant's modesty through needless exposure. If there is evidence that the heart or vascular system is so badly impaired as to make it dangerous, the exercise should be omitted.

Study carefully this sheet so that you are familiar with the specific requirements before you begin your examination.

A. Before exercise

1. Locate the apex of the heart or the point of maximum intensity of the apical impulse and carefully mark its position by (X) on the diagram.—
Is the heart enlarged? ————
Specify degree. ————
Is there a palpable thrill or shock in any of the valve areas? ————
Heart rate at apex. ————
Any arrhythmia? ————
If present, describe. (See B3 of this Form)

2. Is there a murmur present? ————
Is it systolic—diastolic—or presystolic? ————
Mark carefully by ⊙ the point where the murmur is most clearly heard (the point of maximum intensity).
Is the murmur transmitted or heard clearly in any other area? ————
Indicate direction of any transmission by ⟶ .
Is the murmur constant; that is, is it heard in the erect——recumbent position——on full inspiration ——on full expiration? ————

B. After exercise

1. Heart rate at apex immediately after exercise—and two minutes after exercise ————
Count at least 30 seconds on each.
Is the pulse rate at wrist the same as the heart rate at these times? ————

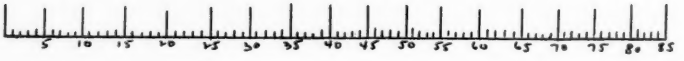
2. Effect of exercise on any murmur present. ————
If the murmur appears after exercise state whether it persists and if it is then transmitted.

3. Rhythm. Does exercise abolish—decrease—or increase the arrhythmia? ————

Count for at least one minute before—and after exercise. ————
Indicate the arrhythmic beats in the lines below

Before exercise

After exercise



C. History

1. Is there a history of acute rheumatic fever? If there is and you have also found a systolic murmur search carefully for any diastolic murmur in the apical area. ————
2. Is there a history of frequent sore throats, streptococcic or other infectious diseases? ————
3. Is there a history of hypertension or "precordial (substernal) pain or pressure?" ————
   Are there any signs of myocardial insufficiency or of coronary artery disease? If there are, describe in detail. ————

* * * * * *

PRESIDENT WENSTRAND—Discussion on this instructive and interesting paper will be opened by Dr. Edward J. Campbell of the New York Life Insurance Company. Dr. Campbell!

DR. CAMPBELL—My discussion of Dr. Bonnett's paper is limited to a comparison of his study with a study made by the New York Life in 1934, covering the issues of 1925 to

1931 carried to the anniversaries in 1933 and reported in the discussion of Dr. Scott's paper reported in the Proceedings of 1934. In our study each application was reviewed by a member of the Medical Board who made his estimate of the actual pathology and was not influenced alone by the opinion of the examiner but depended upon the description of the murmur heard, as well as information from any other source. This is in contrast with Dr. Bonnett's study in which the opinion of the medical examiner was independent of the medical underwriter's appraisal of the impairment. All cases were eliminated in which there was any other factor which might influence our mortality experience. Dr. Bonnett states that they were not dealing with homogeneous groups of accurately diagnosed lesions or at times even with closely related clinical entities, whereas our 1934 study attempted to group only homogeneous material. The difference in results can possibly be largely due to the approach to the problem.

This is the third time I have appeared before this organization and believe I may be excused in continuing to ride my hobby that reliable results are best obtained by the study of material known to be homogeneous. Dr. Hunter mentioned this point at the meeting in Hartford in 1931 when he reported on the MIS of 1929. In the Transactions of the Actuarial Society, in 1932, Edward Marshall reports as follows:

> "The interpretation of mortality statistics is beset with many pitfalls. There are so many inter-playing factors which influence statistical results, and these factors vary so greatly between the different groups studied, that misleading information may easily be deduced from seemingly reliable percentages of actual to expected mortality. Such statistics are receiving particular attention at present, not only because of the recent appearance of the Joint Medical Impairment Study and the Joint Occupation Study, but also because of the higher level of mortality experienced by companies since 1928." And he quotes as one of the important factors "Is the material homogeneous?"

In our paper of 1934 we emphasized the point that in our selection of risks we frequently take into consideration factors which we do not and cannot code for actuarial purposes and hence are not available in a study based on actuarial cards alone.

Apex Murmur Transmitted to Axilla or Mitral Regurgitation Without Hypertrophy

	Metropolitan Issues 1925–1939	New York Life Study Issues 1925–1931 Anniversaries of 1933
All Ages, without hypertrophy	282%	169%
slight hypertrophy	339%	185%
moderate hypertrophy	609%	370%
Experience by Policy Year Duration		
1—5 years	295%	228%
6 years and up	267%	160%
all ages	282%	192%

Both studies indicate that when a mitral regurgitation is accompanied by hypertrophy the mortality experience is markedly increased. In the New York Life study mortality experience shows a substantial improvement with duration. It is possible that the Metropolitan's higher mortality experiences are due to other factors taken into consideration by the selector which were not coded and that our experience is more favorable because the material was carefully selected.

In the group designated as apex murmur, transmitted, with a history of rheumatism or other streptococcus infection, our experience parallels the Metropolitan's experience. However, from age 40 up, our experience is slightly better and I believe is in keeping with clinical opinion that individuals suffering from rheumatic heart disease who survive to age 40, face an improving prognosis each year that they survive. The similarity of results is probably due to the fact that both companies used considerable care in the selection of this type of risk.

Mitral Regurgitation Without Hypertrophy, With a History of Rheumatism

Experience by Age Groups	Metropolitan		New York Life	
		dead		dead
Under 40	550%	141	472%	119
40 and up	335%	58	293%	57
All ages	463%	199	403%	176
Experience by Duration				
1—5 years	495%	88	394%	140
6 and over	441%	111	442%	36
All ages	463%	199	403%	176

Change in relative mortality, by age group, of constant, systolic apex murmur, transmitted to the left, without hypertrophy

MIS Issues of 1909–1927 to Anniversaries of 1928	Metropolitan Issues of 1925-1939 to Anniversaries of Dec. 31, 1941	New York Life Issues of 1925 to 1939 to Anniversaries of 1940	New York Life Issues of 1925 to 1931 to Anniversaries of 1933
Age at Issue			
Under 40 yrs. 248%	386%	303%	273%
40–49 yrs. 190%	189%	167%	196%
50 and up 198%	118%	152%	90%

In future studies, would it not be advisable to have a sample of the material medically revised? If the study of the whole group was materially different from the group medically revised, then steps should be taken to discover the reason for the discrepancy.

It is possible that we may be facing an increasing critical scrutiny of our underwriting practices, not only by applicants for insurance but by our agency force as well. Many applicants object to being considered as belonging to an underaverage group. Their pride leads them to believe that they are the exception to the usual rule and they are often encouraged in this belief by agents and by their family physician. Perhaps we may be faced with the necessity of revising and subdividing

our mortality studies to meet a growing demand of our clients and agency force.

I wish to thank Dr. Wenstrand for the opportunity of discussing this very worthy contribution to the accumulated material concerning apex murmurs. It certainly confirms and parallels previous studies and is added evidence that our underwriting of this type of impairment is on a sound basis.

PRESIDENT WENSTRAND—Dr. J. Hamilton Crawford, who will continue the discussion, was born in Scotland, where he was educated, receiving his medical degree from Edinburgh University. Before leaving for the United States he became a Member of the Royal College of Physicians. Since coming to New York he has held and holds many appointments as cardiologist in prominent hospitals, both in Manhattan and Brooklyn. He is now Professor of Clinical Medicine at Long Island College of Medicine. He is a Fellow of the American College of Physicians and a member of the American Society of Clinical Investigation, and other medical societies. Dr. Crawford!

DR. CRAWFORD—The significance of the systolic murmur has been one of the most controversial points in cardiology since the stethescope was introduced. For many years any murmur heard over the precordium was thought to indicate heart disease. We now know, of course, that this is not true. Later attention became focused on the myocardium and the importance of systolic murmurs became minimized. More recently all factors are being taken into consideration which is the proper view. Nevertheless, it seems to me that the state of the myocardium is more important than the actual murmur, because a good myocardium can overcome fairly well-marked valvular damage, whereas a badly damaged myocardium tends to give way even when the valvular involvement is slight.

The studies which Doctor Bonnett has presented, as well as the other analyses to which he has referred, involving a large number of individuals followed over a period of years to

death, is invaluable to the practicing physician in enabling him to assess the significance of systolic murmurs. The latter are a frequent source of uncertainty especially when they come in the borderline zone. The cardiologist advises the individual, whereas the company deals with the mass. Unquestionably, the average experience is of great value in judging the individual case although it cannot determine the final opinion.

My experience supports entirely the views presented by Doctor Bonnett. It is quite surprising that his conclusions should parallel the experience of cardiologists when it is considered that these applicants were graded by physicians of markedly varying degrees of competence. Unquestionably, cases with definite valvular damage have been included in those listed as inconsequential murmurs, whereas others with no actual heart disease have been rated up. However, the final analysis seems to have reached the correct conclusion. This does not mean that the examination should not be improved as much as possible, because there are undoubtedly many instances in which the individual has been stigmatized without cause. The change in the mortality rate after the enforcement of more rigid examination shows the importance of more thorough study. The ideal in this respect would be to have every applicant in whom the heart is suspected have a complete cardiac examination, including electrocardiogram and x-ray. This is obviously impossible in view of the large number of applicants.

I do not know whether analyses have been made of the cases examined at the Home Offices or by a competent cardiologist when a complete examination has been performed. If they have not, I feel sure that data of the greatest value to practicing cardiologists would be obtained and I believe it might also be of some value to the companies.

As Doctor Bonnett has shown, there is a higher mortality in the younger age groups showing a systolic murmur. This is undoubtedly due to the fact that many of these were cases with rheumatic involvement and, indeed, with more careful investigation might have been proven to be so. Furthermore, in

the older individuals the serious complications of hypertension, arteriosclerosis and nephritis could be more readily excluded by the type of examination given. The presence or absence of hypertrophy seems to me to be of the utmost importance. I do not believe that any case of hypertrophy, whether or not it is accompanied by a murmur, is a good risk. Of course, one must be sure that the heart is enlarged and the only certain method of obtaining this information is by x-ray. When the apex can be palpated, one can have a reasonably good idea on this point but it is important that the examiner should realize that the actual apex is not necessarily the point of maximum pulsation. Furthermore, in many individuals the apex cannot be palpated and resort is had to percussion. In many short-chested persons the heart percusses and sometimes even the apex may be felt beyond the midclavicular line. This may be clearly shown on x-ray to be due to a transverse position of the heart. I think that a valuable addition to the heart chart would be a description of the type of chest.

Apparently the intensity and quality of the murmur is not given much consideration by the companies probably owing to the variability of the reports; yet, these seem to me to be extremely important, far exceeding in value transmission. I am not impressed by the great importance of the latter. Usually the louder the murmur, the further it is transmitted. One of the most important of all heart murmurs, the presystolic, is usually heard over a comparatively limited area. Location, quality and intensity seem to me the most important factors in assessing the systolic murmur, provided one considers only auscultation. Although phonocardiograms can demonstrate murmurs not appreciated by the ear, they can never replace auscultation because they give no information as to quality and intensity, which is so valuable to the experienced observer.

In conclusion, I should again like to emphasize how valuable these statistical analyses, in which large numbers of individuals are involved, are in helping to clear up many disputed points in clinical medicine.

DR. WENSTRAND—We are fortunate to have with us Dr. H. M. Marvin, who is a cardiologist and has very important work to do with the American Heart Association. Dr. Marvin, may we have a few words from you.

DR. MARVIN—Mr. President, Members and Guests of the Association: I am very grateful for the opportunity of listening to this interesting paper of Dr. Bonnett, and for the unexpected privilege of participating in the discussion.

I am sure you will understand that my random comments cannot have the interest or importance of Dr. Crawford's carefully considered discussion, because I knew nothing of what Dr. Bonnett was to say until I heard his paper this morning. If I disagree in one or two relatively minor respects with what Dr. Crawford has said, I am sure he will forgive me because we are friends of many years' standing.

Perhaps it would be not amiss to remind even this audience of physicians that the attitude of the average physician in private practice and that of the insurance doctor towards the systolic murmur differ in many respects. The average physician in private practice, when he first encounters a systolic murmur, is likely to ask himself, "Does this murmur indicate heart disease or does it not?" And, second, "If it is heart disease, is it such as to require treatment or restriction of the activities of the patient?"

The insurance doctor, on the other hand, is usually not asked to express an opinion as to the significance of the murmur, he is not called upon to prescribe treatment, and, most important of all, he seldom has the opportunity of following the patient for a period of years, as the private physician does.

Now, I do not mean to criticize this difference in attitude. I do wish to suggest that it might be kept in mind because I think it has an important bearing upon a widespread impression among doctors, which I believe to be erroneous or at least not supported by adequate evidence, and that impression, which I encounter more and more frequently in recent years, if stated quite briefly and perhaps inadequately, is this: that

a systolic murmur alone is always evidence of heart disease, because insurance figures have shown that applicants accepted with systolic murmurs alone have a much higher mortality figure than do applicants whose hearts present no murmurs at all.

From a practical and realistic standpoint, one must acknowledge that the insurance company is perfectly justified in rejecting applicants who have systolic murmurs as soon as it has been shown that such murmurs are associated with increased mortality. Indeed, they could scarcely continue to write insurance if they ignored such an association. But I seriously question whether the evidence as given justifies the belief that the systolic murmur in these cases is actually due to heart disease, and for that reason, the collection of a large number of applicants with systolic murmurs, a group large enough to be significant statistically, implies the collaboration of a large group of heterogeneous physicians, and I think I may speak without disrespect in reminding you of a fact that is well known to all of you and which has been alluded to by the previous speakers; namely, that many physicians who examine for life insurance companies are men of inadequate training and unsound judgment; many of them are primarily interested in fields other than internal medicine—obstetrics and orthopedics and pediatrics. Many of them have very little interest in the physical examinations and perform them in the most perfunctory way.

I know from long personal experience that many applicants who have been rejected by life insurance companies on the basis of a systolic murmur actually have other signs of heart disease which have been overlooked or ignored. Every year, I see a certain number of patients who come to me because of their rejection, based on systolic murmur alone, and I find that many of these patients, in addition to a systolic murmur, have clear signs of cardiac enlargement, of accentuation of the apical first sound or the pulmonary second sound, or, in many cases, have faint but unmistakable diastolic murmurs also. In other words, these patients have clear signs of heart

disease quite aside from the systolic murmur; yet, in such cases, it is the systolic murmur alone that gets into the statistics. For that reason, I question the validity of these figures with respect to this one point.

I believe that there are only two ways of securing information that will be of very real value. One has already been mentioned by Dr. Crawford. It consists in a careful study of the mortality experience of those people who have been accepted with systolic murmurs, on the basis of painstaking examinations by carefully trained internists or cardiologists, and of applicants who have not only had physical examinations but also x-ray films of the heart and electrocardiograms, and who are known to have no abnormality whatever except a systolic murmur.

The second, and I think the more important way of establishing the real significance of such murmurs, of finally deciding whether they do in fact indicate rheumatic heart disease or are innocent, is to have a careful, prolonged followup study of a selected group of young people who have been subjected to the type of examination I mentioned by cardiologists or carefully trained internists, and who can be followed over a period of many years by these same carefully trained men.

I think it cannot be done by any single man or any single group or any single company. I think it requires the collaboration of scores of internists or groups, with the results correlated and with the study supervised by some central agency. It seems to me that this is a real opportunity for life insurance companies, not only to benefit themselves, but also to display what I think the more enlightened companies are increasingly displaying—a real interest in the public health aspect of medicine. Thank you very much.

PRESIDENT WENSTRAND—A challenge has been thrown in our lap. Perhaps, in the course of time, we may be able to make some more profound studies in regard to the systolic murmur.

I am going to ask Mr. Edward A. Lew to close the discussion. Mr. Lew!

MR. LEW—Mr. President and Gentlemen: With your indulgence, I would like to comment briefly on a few points which occurred to me in connection with Dr. Campbell's remarks.

In reviewing the literature on the subject, we naturally turned to Dr. R. W. Scott's paper on "The Significance of the Systolic Murmur," presented to this Association in 1934. We noted that in discussing this paper, Dr. Campbell had not only pointed to the weakness of the existing classifications of heart murmurs but also went on to demonstrate that some of the broader considerations which underwriters take into account have a very pronounced effect on mortality. Our study reached somewhat similar conclusions from a different approach. We determined the mortality experience of insured lives with systolic heart murmurs as part of a general investigation of medical impairments coded on the basis of the Joint Committee's classifications. We gave some consideration to going back to the original application papers as Dr. Campbell did in 1934, but felt that such a procedure while tending to give more homogeneous data from the underwriting viewpoint, still could not eliminate the fundamental heterogeneity of the material due to the varying significance of the reports made by our examining physicians. Because of this and the desirability of comparing our results with those previously published in the Medical Impairment Study and by Dr. A. Hunter, we decided to make our investigation of the mortality under systolic heart murmurs on the basis of the Joint Committee's classifications.

In view of some of Dr. Campbell's remarks, I believe there may be some misunderstanding as to the significance and comparability of ratios which express the mortality experienced on a particular impairment as a percentage of the Company's average mortality on standard risks during the period of years covered by the investigation. This has been the customary method of indicating the results of a mortality investigation among insured lives with certain characteristics. What is perhaps not adequately realized is that the average mortality of standard risks over a period of years, which is

used as the yardstick for measuring the mortality of lives with different characteristics, varies from investigation to investigation and from one company to another, so that in making comparisons between the mortality ratios obtained in different investigations, allowance must be made for the difference in yardsticks.

The effects of using such different yardsticks are readily demonstrated by reference to the results obtained in the Medical Impairment Study, in the Metropolitan's investigation and the investigation cited by Dr. Campbell.

The Medical Impairment Study covered the issues of 1909 through 1927 carried to anniversaries in 1928 and the yardstick used in that study was the average mortality among standard lives during the years 1909–1928. By way of illustration let us assume that this yardstick at ages under 40 at issue happened to be 5 deaths per 1,000.

The Metropolitan's investigation presented in our paper covered the issues of 1925 through 1939 carried to December 31, 1941. Because of the substantial decrease in the level of mortality at ages under 40, the yardstick used in the Metropolitan's investigation for ages under 40 at issue may be taken as, say, 3½ deaths per 1,000.

The investigation to which Dr. Campbell referred covered, I believe, the issues of 1925 through 1931 carried to anniversaries in 1933. It should be clear that the yardstick used in that investigation, if it represented the average mortality of the New York Life Insurance Company during the period of years covered by the investigation, must at ages under 40 at issue have been somewhere between the 5 deaths per 1,000 assumed for the Medical Impairment Study and the 3½ deaths per 1,000 assumed for the Metropolitan's investigation.

As nearly as we can tell from our investigation and that of New York Life recently published by Dr. Hunter (which covered practically the same period of years as our investigation) the absolute mortality of insured lives with serious heart impairments has not changed materially from that for the period of years covered by the Medical Impairment Study.

This means that persons with serious heart impairments have in recent years experienced about the same number of deaths per 1,000 as during the years covered by the Medical Impairment Study. If for purposes of illustration we assume that the mortality rate among such persons at ages under 40 at issue has been about 12½ per 1,000, then this death rate expressed as a percentage of the yardstick used in the Medical Impairment Study would have given a mortality ratio of 250%. Expressed as a percentage of the yardstick used in the Metropolitan's investigation, it would give a mortality ratio of almost 360%.

A comparison of these mortality ratios does not, of course, furnish any basis for the conclusion that the absolute mortality of persons with serious heart impairments has gone up. All that can be said is that the mortality of such persons has increased in relation to the level of standard mortality (which has decreased). This is another way of saying that in investigations covering recent years we have automatically used different yardsticks from those used in the investigations made in the past.

This difference in yard sticks must be kept in mind in making comparisons between the mortality ratios cited by Dr. Campbell and those in the Metropolitan's investigation, which covered a more recent period of years. Because the yardstick used in Dr. Campbell's investigation was probably at least ½ death per 1,000 greater at ages under 40 than that used in the Metropolitan's study, it automatically produced lower mortality ratios in Dr. Campbell's experience as compared with the Metropolitan's experience. The yardstick used in Dr. Campbell's investigation was somewhat greater not only on account of the higher level of standard mortality prevailing during the years covered by his investigation but also because the level of standard mortality in the New York Life happens to have been slightly higher than that in the Metropolitan even over the same period of years. Moreover in quoting mortality ratios for all ages combined, Dr. Campbell had apparently overlooked that the Metropolitan's experience was

more heavily weighted at the younger ages at issue than the New York Life's. Since the relative mortality of persons with serious heart impairments is highest at the younger ages, the younger age distribution in the Metropolitan experience was also an important factor in producing a higher mortality ratio in the Metropolitan's experience for all ages combined.

The lower mortality ratios quoted by Dr. Campbell do not therefore necessarily imply lower absolute mortality among risks with serious heart impairments insured in the New York Life as compared with similar risks insured in the Metropolitan. Evidence to the effect that the mortality of risks with serious heart impairments has been about the same in both companies is offered by the comparison of the Metropolitan's experience with the closely corresponding experience of the New York Life, recently published by Dr. Hunter (T.A.S.A. XLII, p. 66) and shown in our paper in Table IV. The conclusion that there has been but little change in the absolute mortality of persons with serious heart impairments is, I believe, in accord with medical opinion.

Both Dr. Bonnett and I wish to express our gratitude to Drs. Campbell, Crawford and Marvin for their illuminating remarks. I thank you.

PRESIDENT WENSTRAND—We are happy to have with us this morning Colonel Montgomery Schuyler, who is an Assistant Director of Red Cross Society Relief. Colonel Schuyler, will you say a few words to us.

COLONEL SCHUYLER—I deem it a very great pleasure to be with you gentlemen and to address this distinguished gathering. It so happens that the last time I talked before any insurance group was in this same hotel, several months ago, on the occasion of a very nice luncheon to which I was invited. I was told that the president would see me in just a moment. I turned around, expecting to see a learned gentleman, and I found a charming young lady with very carefully manicured hair. I see that in this assemblage, you haven't quite got to

the point where you have ladies as presidents of your Association.

I am one of the few Americans who does not like to talk before people, and the most cheering note that I heard today was when I came in — that you gentlemen were a little behind schedule — because that makes me think that I can be very short in my remarks this morning, and I probably won't hear any systolic murmurs of discontent over it. Systolic murmurs do add to discontent, or don't they?

The New York Chapter of the American Red Cross, of which I have the honor to be the Acting Director at this time, upon the outbreak of this war put itself on a war footing. For many years, as you know, the Red Cross has attempted to take care of the victims of normal disaster; that is, floods, fires and so on. With the beginning of this war, and under the provisions of its charter from the United States Government of 1905, the American Red Cross is equipped and authorized to take over disaster relief in the various parts of the country where it is necessary.

I am speaking now only for the New York Chapter, which includes Manhattan and the Bronx. There, we have put into effect, under the able guidance of Colonel Earl Booth, who is away for a month or so on a vacation, a system which we have tried and believe will work very well in case of enemy-caused disaster. In other words, we have switched our normal disaster relief in some parts, or in some ways, to enemy disaster relief.

In New York and the Bronx, there are 36 police precincts, and we have divided our relief work up with the control centering in the chapter house at Lexington Avenue and 38th Street, and extending out through a system of precinct organizations through these 36 precincts. We have in each precinct a precinct manager and a certain staff, that I will describe more in detail in a moment.

What happens is that when we get from the control center one of these flashes of blue, yellow or others of these color-

chrome things that you hear about nowadays, everybody at once stands by. The Chairman of the New York Chapter, Colonel Harbord, Executive Director General Robert Davis, the Director of Disaster Relief, Colonel Booth (if he is here) and I go to the chapter house and assume control of this Manhattan and Bronx organization.

Upon notification that there is an incident in a certain precinct, the Director of Disaster Relief proceeds there and joins the precinct manager. They then both go to the scene of the disaster and estimate as nearly and as quickly as possible what the probable amount of disaster is, how many people are going to be rendered homeless, how many people will need food, clothing or other supplies, and what amount of square blocks or whatever proportion of the city may be destroyed or rendered unfit for home use. We then open one of the so-called rest centers, of which we have perhaps ten or fifteen in each police precinct, the assistant manager in charge of the rest center goes there, the nurses go there, and the other personnel, and then we begin to register at that point, which should be within a block or so of the police precinct station house, the people who in one way or another need assistance.

In other words, we are trying to fill the gap between the actual time of the injury and the people being rendered homeless until the city departments get into working order, which is a little bit slower, probably, than we can do. We expect to be on this job within fifteen minutes of the time of the alert, and on the spot within about fifteen minutes of the time of the notification of the damage there.

The precinct organization consists of a manager, five or six assistant managers, three nurses, nine nurses' aids, ten staff assistants, ten precinct aids, twenty Boy Scouts and five motor cars. I also have a detachment there from my city patrol corps — and I should apologize to you for being here in this uniform this morning, which has nothing to do with the Red Cross, but is a New York City Patrol Corps, of which

I am Chief of Staff and executive. We are working very closely with the Red Cross in all disaster work.

Those figures that I have given you are a minimum for the precincts. They can be added to or taken away from, if necessary, at the discretion of the precinct manager. One of the chief things which we have got to do is to provide for lost people, and these staff assistants mentioned here are going to record the names and addresses of all people sent to one of these rest centers. Of course, when we get a blackout or an alarm now, we get an all-clear within a few minutes after it, and then there is nothing much to be done except to assemble and stand by, but in the event of a real disaster, we don't know when the all-clear is going to be sounded; it may be all night, it may be twenty-four hours before these enemy planes that may be passing overhead, or the enemy warships which may be shelling our city, have stopped their destructive work, and we anticipate that a very large number of people, when their homes are destroyed or their families are separated, are going to be rushing around, looking for their fathers, their mothers, their boy friends, their girl friends, or their children.

That is one of the great gaps that we are trying to fill up, and these staff assistants will take the names of all people whom we send to a certain relief center, register them there and when inquiries are made at this precinct headquarters, as will be the case, because they will be directed there by the Boy Scouts and the precinct aids, the greatly worried families are going to be able to find out where their members have been taken.

At the same time that this is being done, I, if I am in charge of headquarters, notify our canteen service immediately to begin getting hot coffee ready, sandwiches, soup, and so forth. This is rushed there in the station wagons and motor trucks that we have, and at the present time, we have about three hundred that are working for us in the New York City Chapter. In addition to that, in the way of food, we have

also made arrangements with the chain restaurants to open their kitchens and immediately begin cooking meals for us; so that we have an almost indefinite supply of food, hot drinks and so on, which can be supplied.

We are not supposed to take over the care of wounded people. That is to be done by the city hospitals. But our stretcher bearers, four hundred or so in number, will probably help the city hospitals and their personnel in getting the people to the hospital, putting the wounded and those unable to care for themselves in the ambulances or in other motor cars, and sending them to the designated hospital.

At this time, I can give you only the roughest kind of sketch of this. It would be a pleasure to be able to stand here and tell you about it for fifteen or twenty minutes, but as I know you are behind time, I am going to confine myself to just these very few minutes, and say that if there are any questions on which I have not made myself clear, I will be glad to answer them. I want you to excuse the hasty method in which I have had to present this to you this morning, because I am really in a great rush. I expect to have some rather urgent things to do before the end of the day, and, consequently, I am going to ask to be excused. I thank you once more for the honor of being present with you.

PRESIDENT WENSTRAND—The next address is one to which we will listen with a geat deal of interest, I am sure. Colonel Eugen G. Reinartz, Commandant, Army School of Aviation Medicine, Randolph Field, Texas, was born and reared at East Liverpool, Ohio. He entered Military Service during the World War in 1917, was commissioned at Fort Benjamin Harrison, Indiana, and made one of the first Flight Surgeons in the Air Corps of the U. S. Army. He has seen twenty-five years' service in aviation medicine, and has served at all the major Air Stations in the U. S., Foreign service in China and Japan, at Corregidor, Ft. Stotsenburg and Nichols Field in the Phillipines, at Pearl Harbor and Hickam Field in Hawaii.

He has served on repeated occasions with the Air Corps Board for the selection of Air Corps Personnel from the graduating classes of the U. S. Military Academy at West Point, and has lectured widely at various colleges and universities on aviation medicine. His chief interest lies in the field of Psychiatry of Aviation, and recently he was made one of the charter members of the American Association of Flight Surgeons. He is a member of the American College of Surgeons. He recently returned from a survey of the schools in the West where Army physicians are being trained in aviation medicine. Colonel Reinartz will address us on "The Effect of Flight on Man." Colonel Reinartz!

THE EFFECT OF FLIGHT ON MAN

By Eugen G. Reinartz,
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Colonel, Medical Corps, U. S. Army
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Since the immortal moment when Daedalus and his son Icarus were made conscious of the melting of wax and for eons before that time, man has watched the flight of birds and has desired to emulate it. However, by reason of being a terrestial animal, this mode of transportation was denied him, at least in the manner in which we now know it, until the successful flights of the Wright brothers, which flights are within the memory of most everyone here present. Man has not been endowed by his Creator with those adaptations which would permit him to fly naturally, as do birds. These creatures do instinctively that which we who would emulate them must do mechanically.

Engineers with their special skills have developed machines for the various purposes for which they are needed but modern aircraft has in many instances outdistanced man's adaptability.

Because flying is foreign to an individual's biological development and not at all in keeping with his natural experiences, it creates stresses and strains that call for rapid adaptation to numerous situations depending on the degree to which man is psychically and physically unspecialized for the task.

It is by reason of these quick adaptations and the stresses and strains incident thereto that the effect of flight on man can be discussed. It is inconceivable that such a discussion should be had in connection with a flight of migratory birds except to wax poetic because of the precision of formation, the unerring exactness of routes travelled and the instinctive reactions manifest.

The art of flying, therefore, is immediately recognized as a difficult one to master, certainly when viewed from the military standpoint. As difficulties develop, they naturally fall into the medical field and medicine has been most closely linked with aviation since its inception.

At the present time upwards of 2,000,000 individuals fly each year, and the time has come when every practicing physician must of necessity have knowledge concerning the effect of flight on the human economy and be prepared to advise intelligently, treat or examine those interested in flying, from whatever standpoint. Every physician who is properly trained must now also be trained in facts concerning aviation medicine just as he is trained in other specialties of medicine.

The detailing of the history of modern aviation medicine, in brief, is deemed essential, as there may be those among you to whom this branch of medicine is a name only. In 1910, the Germans drew up minimum physical standards for their military aviators, and by 1915, the medical control of aviators was a reality. The French did nothing much about this branch of medicine until 1915; the English, not until 1917.

During the early years of World War I, it was determined that 90 per cent of the fatal accidents were due to the pilot himself, and this sharply focused the attention of military authorities on the medical problem associated with flying. By the end of 1917, all European armies had a medical department as an integral part of their flying services. The first mention made concerning aviation medicine in the United States was by the Army in the early winter of 1912. In 1918, following the death of Major Ream, M. C., the first medical officer to be killed in a "crash," the word "Flight Surgeon" was coined.

In 1919, the Air Service Medical Research Laboratory was established and this through the years has developed into what is now the School of Aviation Medicine, at Randolph Field, Texas. The character of the School changed, however,

and from a purely research laboratory, a teaching school for Flight Surgeons developed. In 1926, a Flight Surgeon collaborating with an Air Corps officer brought out the correlation between the illusions of the senses as related to the internal ear and the number of crashes that had been occurring. This, in the opinion of the writer, next to flying itself, is the most important contribution to modern aviation. It was this correlation that has been responsible for the tremendous upswing in aviation. It has been responsible for the completion of every flight when so-called "blind flying" conditions are encountered.

In January, 1937, a new aero medical research laboratory began functioning at the Materiel Division at Dayton, Ohio, and a new chapter in aviation research was begun. This laboratory has latterly occupied itself almost solely with the so-called "occupational" diseases that beset the pilot while engaged in flying. In 1940, the Navy established a School of Aviation Medicine at its training station at Pensacola, Florida. In January of this year, a research section of the School of Aviation Medicine was established. This has for its goal the solution of problems concerned with the selection of applicants for aviation training and for a determination if possible, as to what physical and psychological characteristics are necessary for those who would aspire to become military aviators. The history of aviation medicine is so closely linked with aviation itself that it is impossible to tell the story of one without, at least in part, giving the history of the other. This is produced by the very intimate relationship between certain basic principles of aviation medicine and success in the design, operation and piloting of aircraft.

Practically all improvements made in airplanes except those involved in aero-dynamics and motors, have in their entirety or in part been due to the activities of the Flight Surgeon or medical men in connection with aviation.

Before discussing some of the effects of flight on man, and since the Flight Surgeon is assuming such an important place

in the war effort and his duties are bulking large in the ever expanding Army Air Forces, it may be well to speak of him, through whom the impetus, for the modern advances in aviation medicine, was given. He must have a solid foundation in general medicine complemented by a large fund of special knowledge. So equipped, he will have poise and dignity and his judgments will carry weight and his opinions will be respected. Furthermore, a Flight Surgeon, so implemented will inspire confidence in flying personnel whom he has to examine. He must be free from prejudice and show no favoritism. He must be courageous and be prepared to defend his opinions with facts and logic. His loyalty and integrity must be unquestioned. His understanding of human nature must be profound so that those with whom he associates will instinctively turn to him for guidance and advice. A good Flight Surgeon, therefore, must be a combination of an outstanding physician, preacher and lawyer with a strong leaning toward the psychopathological side of medicine.

The duties of the Flight Surgeon fall into four categories. First, he must be an outstanding physician so that he may properly care for such of his personnel who become ill; his is the responsibility of selecting candidates for flying training subsequent to their arrival at an Army Air Forces Classification Center; his principal duty is the "care of the flyer," and last, he must continually investigate the effect of flight and seek remedies for those factors that have deleterious effects upon flying personnel.

Thus it is seen that the difficult situation can be solved only by those who endeavor to solve satisfactorily the "art" of aviation medicine.

Aviation medicine has much to offer a properly qualified physician, since there are but few in civil life who are trained for such work or who are prepared to devote their lives to this work.

The selection physically of those who will eventually be assigned to pilot's training will be of particular interest to a

group such as this whose entire existence revolves about physical standards, physical fitness, longevity and other actuarial data.

Vision and eye muscle balance must be excellent, because it is the eye and its cerebral correlations that is the most important single organ in flight and the one through which external reference is maintained. Depth perception must be determined, as this is brought into play with every take-off and landing. The improper estimation of distance in a rapidly moving plane or in combat has in many cases had fatal consequences. Color vision is designated as safe or unsafe and is tested with the American Optical Company pseudo-isochromatic charts and the S.A.M. lantern. Eye grounds are searched and refraction is performed on each cadet applicant whose examination would indicate that he had a possible latent defect.

An exhaustive examination is made of the nose and throat with the view to discovering any condition that might lead to the development of the condition known as aero-sinusitis. Air passages must be free and any deviation from the normal must be corrected.

The ears are thoroughly investigated and the patency of the eustachian tube is tested due to the fact that difficulties may arise by reason of the changing of barometric pressures in ascent or descent giving rise to a condition of aero-otitis. Hearing, of course, must be normal because of the necessity of using radio.

The general physical examination is one of the most rigid given anywhere or for any reason, a $100,000 life insurance policy, not excepted. A stereoscopic x-ray of the chest, blood serology, a urine examination and stethography are routine with such other investigations as electrocardiography, electroencephalography, gastric analyses, blood chemistry, x-rays of organs or bones, kidney function tests, etc. made in those cases where any or all of those tests are indicated. All laboratory aids to diagnosis are available and are used. We believe

the physical examination given an aviation cadet to be one of the most comprehensive routinely made.

The Schneider Index is still used but its efficacy as a test of physical fitness is doubted. Since the pulse and blood pressure are taken under varying conditions and these may be an index to the emotional tension of the examinee, this test is used most frequently for the information it gives to the examiner as to one phase of the candidate's psychic make-up.

It is evident that an individual may fly in spite of some physical handicap, however, it is inconceivable for an individual to fly with a marked handicap of psychogenic origin, a loss of stability or an improper coordination of mind and body.

In connection with the regular physical examination, there was also conducted, in the past, a comprehensive mental examination given for a determination of the candidate's psychic make-up. It took into consideration those characteristics that make for instability or stability and if the latter were in preponderance the man was qualified. However, if he showed an unstable make-up, he was disqualified. Careful investigation was made into any evidence of the epilepsies, epileptic equivalents and an appraisal of his epistatic overlay. The psychic examination of the past consumed about four hours for its proper performance. This in peace times was no great problem. However, in war no such exhaustive examination could be made and new rapid tests had to be evolved. An applicant now is given what is known as a psychological "screening" test when he is first enlisted. Should he pass this test, he is classified for acceptance for training as a member of an air crew. He is then given a battery of "paper and pencil" tests in which practical affairs are emphasized and from which it is then determined to which type of duty he is to be sent. The test includes tests for practical judgment, arithmetic, reasoning, vocabulary, mechanical comprehension, reading comprehension and knowledge of current affairs. To these are added a battery of psychomotor tests. These tests are

given for the purpose of classifying air crew applicants into pilots, bombardiers and navigators. The tests while evaluating certain phases of the cadet applicant's make-up have failed in their evaluation of the individual as a functioning whole so that it has been found necessary to reinstitute the neuropsychic examination in abbreviated form, so that a fully rounded picture of the applicant's personality may be had.

Mundanity in the past has determined the activity of man to be confined to a two dimensional activity. Comparative slow speeds have been his personal lot through the ages; climatically he has been able to adapt himself to temperatures of from 35° to 0° C; the atmosphere he breathed was approximately 21% oxygen, under an atmospheric pressure of from 650 mm. Hg. to sea level pressures of 760 mm. Hg.; he was restricted in his movements and developed an immunity to the various vicissitudes of his immediate environment. This all has been changed by the advent of modern flying.

It must again be stressed that with the advent of flying, as we now know it, tremendous stresses and strains have been placed on this terrestial animal called man. Operational speeds have been stepped up to over 400 m.p.h., operational ceilings—the altitude to which man may fly—have increased to 35 or 40,000 ft. where atmospheric pressures are reduced to approximately one fifth of ground level pressures; gravitational forces, both positive and negative have been brought into play that never had been dreamed of in their application to the human body.

The air, which forms an envelope about the earth's surface, decreases in density with altitude, consequently the pressure at which oxygen is delivered to the lungs and therefore to the tissues is correspondingly less. This creates a condition called anoxia. Anoxia becomes critical at altitudes between 15 and 20,000 ft. varying with the physical condition of the individual, but under no circumstances can an individual function entirely normally under these lowered oxygen tensions above 20,000 ft. The symptoms of anoxia are increased

respiratory and pulse rate, cyanosis, muscular and mental incoordination with a loss of memory. If continued the condition ends with collapse. It must be remembered that the development of symptoms is insidious and these are often present in severe degree in spite of a subjective exaggerated sense of well being. A false sense of security is produced and the condition is not unlike that of inebriation of a moderate degree.

The normal percentage of saturation of the blood with oxygen, which is 96%, is necessary for a normally functioning nervous system. Any deviation from this normal, even as little as to 90%, affects it appreciably and consciousness is lost at about 25 to 30% reduction. By the use of supplementary oxygen, this diminished saturation can be brought up to and above 93% and held there permitting normal function, within the altitude range of normal operations.

A far reaching experimental work has been reported by a Flight Surgeon on the "Effects of Repeated Anoxia on the Brain(1)." This oxygen deprivation of the brain has assumed great practical significance in recent years, and more particularly since the air war, at almost unheard of altitudes, has assumed such large proportions. In order to function properly, the nervous system must have an adequate supply of oxygen. The experiments would seem to indicate that no prolonged periods of significant oxygen want leave the brain totally unscathed.

The most practicable method of prevention of anoxia is the giving of an increasing amount of oxygen in consonance with the greater need, as higher altitudes are reached. A second means is by the use of the pressurized airplane cabin.

In the past, military pilots have been reluctant to use oxygen because they believed it a reflection on their ability to ascend to an altitude, because it was disagreeable to use with the equipment then available and because they thought its use personally harmful. This, with education and improvement of equipment, is no longer the case. If an individual

has a low altitude tolerance, he must of necessity begin using oxygen sooner than the individual whose tolerance is greater. The amount of oxygen administered is that amount required to maintain a normal sea level pressure of oxygen in the lungs plus a small excess as a margin of safety for unforeseen circumstances and emergencies.

Civilian airlines have recently introduced the use of oxygen on certain "runs." When a "run" is to be above 10,000 feet for one-half hour or more, the use of oxygen by crew and passengers alike, is mandatory.

The attitude of the average passenger concerning the inhalation of oxygen will vary through the whole gamut from that of experiencing a novelty to that of experiencing a morbid dread.

The oxygen mask used in civilian airlines permits eating and speaking. However, the use of any device for the administration of oxygen confines the passenger to his seat or in its immediate vicinity. The disadvantages of the mask on a sleeper plane can be readily visualized, unless it is intended that the passenger remain awake.

Within the recent past, certain airline companies have placed in operation "pressurized" airplanes whereby it is possible to fly at 15 to 20,000 feet in perfect comfort without the annoyance of oxygen or the equipment for its administration.

Such pressurized cabins are now being experimented with in the Army. While it would seem that a sealed cabin and enemy gun fire are inconsistent and incompatible, nevertheless, the compressors used are of such strength as to be able to maintain, in not too acute an emergency, the increased pressure, at least partially, until lower altitudes could be reached where pressurizing the cabin would no longer be needed. These statements, of course, do not apply where the cabin of the airplane is riddled with numerous bullet holes or torn apart by a cannon shot or shrapnel.

It is axiomatic that a pilot must have good vision to orient himself and his plane in a three dimensional space.

The eyes of the pilot are particularly subject to the forces that are brought to bear and at times these approach the endurance load. When one visualizes the strain incident to hours of flying in the dazzling glare of the ocean, over deserts, glaciers, and clouds by day and the assault of the visual mechanism by searchlights by night, one gets an idea of the stress that is placed upon these organs. This affects particularly the fusion ability. In actual combat, the pilot must make rapid and correct decisions based on his visual impressions, if by day, then primarily by photopic or "cone" vision and if in the dark of the night, then primarily by his scotopic or "rod" visual apparatus. This difference of day and night vision was the subject of an illuminating article in the magazine LIFE of October 5, 1942.

The finding of the enemy target at night before himself being a target, and night landings when exhausted, are some of the difficulties encountered by the pilot and their solution is dependent on his acuity of night vision.

During aerial combat maneuvers either in training or under actual battle conditions, great stresses are applied directly to the physical structure of the body, body tissues and fluids of flying personnel, due to acceleration, deceleration and centrifugal forces. These forces are measured in terms of gravitational units, the so-called "G". One "G" represents the force of the static weight of a substance or combination of substances. One's indicated weight is one's "G". If one stood on a scale in an ascending elevator, that started rapidly, the scale might show one's weight to be twice as much as the static weight. This would represent 2 "Gs". After the elevator had reached a constant speed, the indicator would return to a reading of one's true weight or one "G". On the contrary, were the elevator to decelerate, the indicated weight would be less and this would be stated then as negative "G". Thus it is seen that the body is influenced by acceleration or de-

celeration but that constant linear motion regardless of its velocity produces no effect. This positive "G" is experienced by military flyers under conditions where the airplane might be catapulted from a battleship. Changes in direction of motion bring into play centrifugal forces which affect the body of the flyer similarly, positively or negatively. This depends on the direction of the maneuver — dive bombing, snaprolls or an inside loop creating positive forces and an outside loop creating negative forces to be exerted.

The most spectacular of the phenomena resulting from the application of these forces is the so-called "blackout" or if less severe, the "grayout." The former is of common occurrence in sharp pull-outs after a dive and the latter may be experienced in pull-outs not so severe or even when flying at great speeds in rough air.

The exact cause of this condition is controversial though it is generally accepted that it is due to retinal and cerebral anemia that is produced when blood is shifted from the cerebral hemispheres to the splanchnic area or extremities. A healthy young pilot can tolerate a force of from 4½ to 5 Gs for a short period without blackout.

Attempts at overcoming these forces have been made by the use of various types of anti G suits, cervical collars and devices such as the Germans use in which the pilot's seat is adjustable and just at the point of pull-out the pilot is in an almost supine position. By crouching down and raising the level of the lower extremities, it is also possible to increase tolerance to this force.

Lately, due to the marked increase in the rapidity with which ascents can be made and high altitude reached, attention has been directed to the alterations in pressure of another of the components of air — namely, nitrogen.

The formation of nitrogen bubbles in the body at high altitudes is fundamentally the same physical process that causes them to appear in deep sea divers and in workers in compressed air — the so-called "caisson disease" or "bends."

This condition as applied to aeronautics is known as aeroembolism. It is a disease produced by a rapid decrease of pressure below one atmosphere, such as may occur in aircraft flights to high altitude and which is marked by the formation of nitrogen bubbles in the body tissues and fluids.

The body tissues and fluids are saturated with atmospheric gases at sea level pressure since the blood in the lungs is exposed to these gases and dissolves them each according to their partial pressures. The blood of the lungs takes up in simple solution relatively large amounts of nitrogen while oxygen and carbon dioxide are taken up in lesser amounts. While most of the oxygen is consumed by the body tissues, the nitrogen is inert and is not utilized but goes into simple solution in an amount dependent on the partial pressure of the nitrogen in the lungs. Thus at sea level pressure, the body tissues are always completely saturated with atmospheric nitrogen.

During ascent in aircraft, the internal partial pressure of the body nitrogen is above that of the nitrogen of the lungs and the tissues are therefore supersaturated. As a consequence, the nitrogen dissolved in the blood begins to be given off in the lungs; the nitrogen in the tissue begins to enter the blood stream and in this way the body tends to rid itself of nitrogen. If the ascent is slow, elimination of nitrogen takes place in this manner, but if rapid, the nitrogen will come out of solution and form bubbles. These bubbles form not only in the blood, but also in the other tissues of the body. During ascent, bubbles first appear in the spinal fluid, this is because of the poorer elimination of nitrogen from this tissue, than from the blood or other tissues.

In view of the fact that the service ceiling of many of our aircraft is now 35,000 feet and the necessity of reaching that ceiling in the least possible time, especially in military aircraft, is imperative, it is readily realized that ascents of more than 1000 feet per minute, would not be unusual. This is particularly true of the interceptor type of aircraft.

The symptomatology of this disease is dependent, of course, on the portion of the anatomy in which the bubbles of nitrogen lodge. In the spinal fluid, they cause increased intracranial pressure with attendant symptoms. In the tendons, fascia, periosteum, nerve sheaths and bone they cause pain.

Nerve pain may be in the form of a descending neuritis with marked tenderness along the course of the whole nerve, or it may be only peripheral, affecting the nerve endings. Urticarial wheals of large proportions have been noted on the torso and are said to be due to the mechanical irritation of the posterior horn cells of the spinal cord.

In the cardiovascular system, the greatest danger is that of embolism of an end artery in the cerebro-spinal system. This may lead to infarction and result in a catastrophe. Embolism of the pulmonary or coronary vessels may cause edema of the lungs or cardiac failure.

In the lungs, many aeroemboli occur as they are carried there in the blood stream. The lungs act as a strainer and as bubbles collect, more and more vessels of the lungs become blocked and occasionally a large vessel becomes obstructed. The former condition is manifested by a burning sensation, while the latter is manifested by a sharp stabbing pain. This condition is called "the chokes." Edema rapidly develops. However, the rarified atmosphere in which this condition develops will not carry the accumulating serum out of the lungs, so the cough that develops is unproductive until the condition can be relieved by descent to sea level and normal atmospheric pressures.

The most frequent site of pain is around one or more joints and this is usually the first indication of an impending attack of aeroembolism. This pain may be mild at first, but rapidly becomes almost intolerable.

Itching, rashes, pruritus and thermal sensations of hot and cold have been described in connection with this disease. These, it is believed, are due to bubbles forming in or under

the skin and sweat glands. Formications, too, have been frequently noted.

The prophylactic treatment of this condition is, of course, the restriction of flying below levels at which this disease is likely to occur. This is indefinite, but since in experimental animals aeroembolism is known to occur at 18,000 feet, it is believed passengers on commercial airlines should not be flown above this altitude in unsealed cabin ships.

In military flying, it would seem advisable not to fly above 30,000 feet except in rare instances, and then only after special physical examination, instruction in the cause and effects of aeroembolism and where practical, subjected to nitrogen desaturation.

The active treatment of this disease consists of recompression and this in aviation involves only descent to lower altitudes and higher atmospheric pressure. The descent should be started as soon as possible and as rapidly as is consistent with safety. If the attack is severe, descent to sea level or as near sea level as possible, may be necessary. One hundred per cent oxygen should be administered as long as there is evidence of the disease.

Barometric pressure change, coincidental with altitude, affects all closed cavities of the body. All gases undergo relative expansion during ascent and relative contraction during descent. If gases are unrestricted in their expansion, their volume would be doubled at 18,000 feet; quadrupled at 33,000 and expanded eight times their original volume at 48,000 feet. The most important cavities thus affected are the intestinal tract, the middle ear and the paranasal sinuses.

The middle ear and its appendages, for all practical considerations, is a closed cavity except during the act of swallowing, screaming, yawning or other similar maneuvers. During ascent, the relative pressure in the middle ear cavity increases and forces the ear drum out until some maneuver of the pharyngeal muscles opens the eustachian tube and the

pressure is equalized. On descent, the increasing air pressure on the outside, with a relative negative pressure behind the drum, forces this membrane in. As long as swallowing and consequent equalization of pressure take place, no pathological change occurs. It is for the purpose of creating more frequent swallowing and therefore better aeration of the middle ear that airline hostesses offer passengers chewing gum before the descent for a landing is made.

If, however, there is a blockage of the eustachian tube because of regional inflammation, edema, tumors or adenoid tissue, inability to properly equalize the pressure in the middle ear cavity causes a condition known as aero-otitis media. The most serious portion of this problem is in descent as the negative pressure draws the walls of the eustachian tube together and a locked flutter valve is formed. If this lock is not broken, much damage to the ear drum may occur even to rupture. This is why people with congestive conditions of the nose and throat should be most careful when engaging in air travel while so afflicted.

The paranasal sinuses with their small ostia are similarly affected by the conditions enumerated and when so affected, the condition is known as aero-sinusitis. Under normal circumstances, the sinuses drain well. However, when the turbinates and other tissues of the nose are congested, coincident with upper respiratory infection or allergy, the ostia become occluded and a closed cavity is formed. Descending from altitudes, these cavities participate in the same conditions, as does the middle ear with the development of pain. The pain engendered at times is such as to make the pilot believe that the landing of his plane is impossible.

Similarly, gases may be trapped in various portions of the intestinal tract. Expansion of these gases may cause severe "cramps," and producing upward pressure on the diaphragm may cause an embarrassment of respiration, with a limitation of the respiratory excursion thereby deepening an already existing anoxia.

Airsickness while ignored by many from a purely medical aspect and made the object of ridicule, is nevertheless a major problem in aviation.

Candidates for flying training who give a history of seasickness, car sickness, train or swing sickness usually develop airsickness in training. While airsickness does occur among older pilots, it is a rare finding. Passengers and members of the airplane crew are more susceptible than the pilot who is actually at the controls.

The traveling public, being entirely unselected, is much more susceptible to airsickness than personnel whose duty is flying.

Airsickness is not only one of the most far reaching but also one of the most important unsolved problems of aviation medicine.

Two well known facts determine that the etiology of airsickness lies principally in motion. First, the frequency and severity of airsickness, when encountered, are in direct proportion to the amount and duration of the acceleration encountered in flight, and second, that persons with non-functioning or dead labyrinths seldom, if ever, become airsick. It is the opinion of the writer that there is a very close relationship between airsickness and instinctive fear, although this is difficult of proof. This, it is believed, is based on the loss of sustentation and the fear of falling; loss of equilibrium, loss of orientation, or all combined stimulating the self-preservative instinct and giving rise to profound bodily and mental reactions.

The rate of development of symptoms in those about to become airsick depends on the emotional state of the individual and on the amount and duration of acceleration.

The prophylactic treatment for airsickness, as in seasickness, has taxed the armamentarium of Flight Surgeons and airline surgeons. Persons who, by reason of age or debility, are suffering from conditions that may be made worse by

vomiting, cause an increase of heart beat, or an increase in blood pressure should be warned against flying. Neurotics and unstable individuals, who are always present in the flying public, will, no doubt, be airsick even though the flight is made in perfectly smooth air.

A mind free from anxiety and grief is a good prophylaxis. Bad odors, disgusting sights of others vomiting and stuffy air are bad omens for those subject to airsickness. The avoidance of rough turbulent air or the violent maneuvering of the airplane are practical considerations that will tend to keep airsickness at a minimum. Gastric disturbances, especially those due to an overindulgence in alcoholics, indiscretions in diet, or the use of cathartics are particularly prone to cause air passengers to develop this condition. Empty stomachs too, should be avoided and experience has shown that a bland meal, lightly indulged in, is well tolerated.

Just as in seasickness, there is no known remedy for airsickness. The disease is self-limited and has not been known to cause more than a temporary, though distressing disability.

The stimulation of the adrenals and secondarily, the stimulation of the involuntary vegetative sympathetic nervous system are of importance and interest. The stimulation of the adrenals, in respect to flying, is on a basis of fear and anoxia. Fear is one of the most easily and constantly evoked of the biological reactions, since it is the emotion which appears whenever the individual's life or activities are threatened in one way or another. When so jeopardized the reactions of stimulation express themselves in tensing of the muscles, quickened respiration with hyperventilation; marked quickening of the pulse and profuse perspiration. There are numerous occasions while flying, where fear in some form or another is experienced. This is especially true in combat flying and at the same time furnishes the necessary stimulation to carry on the emergency effort. If the effort is long continued, fatigue sets in and fatigue creates a fear of incapacity, fear of failure, and creates a fear of inferiority. Depression

flourishes in the soil of fatigue. A pilot who has spent a sleepless night arises with a feeling of exhaustion, lacks initiative and does not care to fly.

It must be remembered that it is only common sense to be afraid of real danger. It is the problem of the Flight Surgeon to assist in the inculcation of self-discipline and to elucidate to flyers the facts of the situations in which they will find themselves. Real knowledge is one of the best antidotes for unreal fear.

When flying at extreme altitudes surrounded by a harsh environment, the pilot becomes lonely. Loneliness, too, engenders fear. Being gregarious animals, pilots crave companionship under these circumstances. A newspaper report has been received recently of a captured German Ju88 in which the crew positions were extremely close in a narrow cabin. This closeness it was stated was for the purpose of "maintaining morale."

It must ever be remembered that fear is the emotion reserved for the unknown. When a pilot takes his ship to 35 or 40,000 feet, he is certainly in an unknown environment. Fear is also identified with the inscrutable. However, no matter how terrible an impending event, if its occurrence is recognized as a certainty, individuals will react to it with a response other than fear, as do pilots who have been properly trained.

It is a matter of common knowledge that very loud sounds produce sensations of discomfort, at times amounting to acute pain and with persistent ringing in the ears. Long continued exposure to noise of considerable intensity is a cause of deafness. It is estimated that the volume of noise in a closed airplane cockpit amounts to 100 db.

Any factor, such as noise, which tends to lower the physiological level of activity of an organ is especially undesirable in individuals who are exposed to anoxia.

McCartney in an article on "Noise Drives us Crazy(2)," states: "Noise may lead to deafness, but of all hazards, the

effect on the nervous system is the most serious. It lowers all of our faculties. It slackens and dulls our mental processes. It clouds judgment and reduces precision of action. It decreases general efficiency and makes one irritable, pessimistic and grumpy." Just the type of reactions that we are striving by research to eliminate from the occupational hazards of the pilot.

The effect of cold on flying personnel has been the subject of much study.

The average temperature at ground level is approximately 15° C and falls about 2° for every 1000 feet ascent until a minimum temperature of –55° C is reached. With all the research that has been made in the matter of flying equipment, it is not possible to keep warm in the low temperatures encountered at high altitudes. At extreme altitudes, these low temperatures are incompatible with life except for very short periods of time. The protection offered by flying clothing is practically lost at temperatures ranging below –10° F. All manner of symptoms arise, beginning with sensation of chilliness and ranging to actual pain. The hands and the feet are the first to be affected by the cold. This brings with it clumsy movements. The finesse of touch, the diacritical sense, so necessary in an airplane, becomes less acute. Heavy flying clothing, that is not effective, adds to a pilot's burdens not only because of its additional weight, but because it in itself handicaps his movements. Some of the most serious effects of cold upon the pilot are those in the psychic field and are due to the physical discomforts experienced. These bring in their train mental distress, loss of self-confidence, the feeling of "is it worth the price"; and an overpowering desire for the discontinuance of the mission. If the pilot is subjected to such an environment, beyond this point, he will be the subject of an overpowering lethargy that will steal over him quietly, quickly, though certainly, and that will end in stupor and death.

Vibration generally creates tenseness and muscle fatigue.

This may be due to the action directly on the muscles and bony framework or may be due to the mental effect produced by vibration with its secondary tensing of the musculature. Whatever the reason, fatigue results.

The constant vibration experienced in an airplane with its added sensation of instability brings into play again the self-preservative instinct, unconsciously. Where there is vibration, one braces oneself to neutralize its effects. However, when this cannot be overcome and sustentation is jeopardized, the mental forces of the pilot are again brought into conflict with his environment.

For years Flight Surgeons have known of a slowly developing chronic condition occurring in aviators known as "aeroneurosis."

The principal exciting etiological factor in this disease is emotional stress. Pilots, early in their career, realize that by reason of increasing age, minor accidents, and disease, their careers are in jeopardy.

The first change that takes place in a pilot is when he shows minor physical defects which suspend him from flying temporarily. He begins to realize that physical perfection is no longer a gift but something that from that time on must be maintained. A second change that comes about is when he marries and assumes the responsibility of a home and family. The conservatism of age adds to his problems, for now he has more at stake and must protect and provide for others as well.

There are few occupations which so frequently subject those engaged to such marked shocks as does aviation. Serious airplane accidents are the common lot of pilots of years of flying experience, and even though there be no physical injury, a crash invariably produces profound psychic shock. The total effects cannot accurately be evaluated but there can be no doubt of their effect on the central nervous system. The witnessing of crashes and death of one's friends in the most violent type of death known, often complicated by a seething

inferno of gasoline flames, in which the victim writhes in his death agonies, leaves a searing, deep, lasting, emotional scar and gives actual physical pain. Such an experience is not soon forgotten, and yet, in order to fly, such experiences must be repressed and in the repression, if not entirely successful, new conflicts arise.

In flying, the self-preservative instinct is continually called into play. This is one of the most deep seated of instincts and when stimulated may, and frequently does, arouse profound emotional disturbances. The instant one leaves the ground, this instinct is brought into play, as the individual is then projected into a new medium and the sustentation to which he has been accustomed, by reason of being a terrestial animal, is removed. Even though the fear engendered is repressed, it is the opinion of the writer that each pilot experiences the subconscious nervous trauma with its attendant emotional factor each time he goes into the air. Furthermore, it is believed the effects of such trauma are cumulative and have their distinct bearing on the development of later aeroneurosis among those not so well equipped as their fellows to withstand these traumata.

The very nature of a pilot's duties is such as to carry with it all the elements producing mental fatigue—the principal and all important factor is again mental conflict. The concentration and attention constantly required in flying are probably greater than in any other occupation, One needs only to watch the beginning students of flying or flyers of longer experience after a long or arduous mission, to see the exhaustion that is attendant, and the over-powering desire for sleep.

The onset of this condition is insidious and no specific time for its beginning can be set. Many cases of incipient aeroneurosis are detectable by a Flight Surgeon when flying with his borderline cases. In this way, the disease can often be detected weeks before it would otherwise be recognizable.

The prophylactic treatment is one of the greatest problems

for medical men associated with flying and should be an all consuming one so that but few will reach the point where active treatment must be instituted.

During the present war, there is found developing a syndrome known as "flying stress," "flying fatigue," or "operational fatigue." While the aeroneuroses are found in normal individuals who are subjected to stresses and strains over a long period they are not necessarily entirely due to flying as indicated previously. They have their most fertile field in those unstable individuals who have adjusted sufficiently to meet the normal vicissitudes of life, but who retreat in the face of stresses which to them are overwhelming. It is the exceptional individual who is involved. On the contrary, it would seem that "flying stress" or "operational fatigue" is used to describe a condition that may be observed as an *abnormal* flying strain being placed on a *normal* individual. It is particularly found in those members engaged in battle flying. Let me repeat its manifestations are the effect of intense flying upon the normal individual. It is seen in both pilots and air crews alike and appears frequently simultaneously. These well coordinated individuals, presumably because of the care used in their selection for specific assignments and free from psychogenic disorders, can break only on exposure to overwhelming stresses. The assumption is, that at some level of pressure, the most firmly integrated individual will succumb. The characteristics of "operational fatigue" are varied and various but in every case there are sufficient symptoms and signs, no matter how they may be grouped, to arrive at a diagnosis.

It is one of those conditions in which one makes the diagnosis by awareness of the symptom complex and by making an evaluation of the situation, as a whole, instead of by the detection of individual symptoms. For this reason, the total behavior changing somewhat, the squadron commander will most likely be aware of the condition as quickly as will the medical officer. The liaison, therefore, between the com-

mander and the surgeon must be close if cases are to be recognized early.

The condition is characterized by symptoms that begin slowly, but once having begun, develop rapidly with deterioration and disintegration of the mental and physical constitution of the victim. They "crack up" so to speak. It is the sum total of symptoms raising themselves above the diagnostic threshold that brings the individual to the attention of medical personnel. It is the duty of the doctor, therefore, to reduce this diagnostic threshold to the lowest level.

This condition could easily be missed by an observer unless he was well and intimately associated with his pilots. The symptoms are furthermore not such as to bring the patient to the doctor and certainly not in the early stages. Pilots and crews with a strong sense of duty will hide their disability and will not report sick until they are definitely ill, thereby making it difficult to detect their early symptoms for the application of remedial measures. The doctor usually has to find the patient and then unfortunately, it is often too late for rehabilitation and the victim may be lost to his flight, if not permanently, certainly for a period of many months.

As the case progresses, the appearance of the patient is characteristic. His posture is apathetic; his shoulders droop, face is pallid and lined with shadows under the eyes. Mobility is markedly lost. No movement is made unless it is entirely necessary. The condition has been described as though the individual has been "pithed." It might almost be likened to beginning encephalitis lethargica although the patient frequently states that he "feels all right." The blood pressure shows a reduction of from 10 to 20 points below that which has been the individual's normal. The heart action shows some irregularity following exertion. Prophylactically, much can be done and is being done. However, it is not possible to reveal the nature and length of treatment for obvious reasons.

As must have been apparent from the description of some

of the vicissitudes which beset the pilot from the instant he opens his throttle for "take-off," throughout his flight and until he has landed, rolled up to the flying line and "cut his switches," he has been in an element that is baleful, malevolent, harsh, sinister, relentless and destructive. Everything that he must of necessity do to maintain his equilibrium, maintain contact with his environment, complete his mission, and bring his plane safely to port must be done correctly. In many instances, there is no time for afterthought or second choice. Life is continually in jeopardy and the pilot, not adapted by nature to empyreal existence, is continually faced with dissolution.

It is believed that it has been possible to touch some of the high points in the effect flying has on man and give you insight into some of the problems that confront the Flight Surgeon in these stirring and dangerous times.

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PRESIDENT WENSTRAND—Gentlemen, this has opened up a vista, if I may speak figuratively, such as we have not expected. I know that there are many things that we should like to ask Colonel Reinartz, but he assures me that he is going to be with us more or less of the day and this evening, so probably questions may be asked of him in private rather than in public.

The next paper is by Dr. Ernest J. Dewees, Medical Director, and Dr. Paul H. Langner, Jr., Assistant Medical Director of the Provident Mutual Life Insurance Company. Dr. Dewees will read the paper entitled "A Study of Glucose Tolerance Tests and the Significance of Glycosuria." Dr. Dewees!

A STUDY OF GLUCOSE TOLERANCE TESTS

and

THE SIGNIFICANCE OF GLYCOSURIA

By Ernest J. Dewees, M. D.
MEDICAL DIRECTOR

AND

Paul H. Langner, Jr., M. D.
ASSISTANT MEDICAL DIRECTOR

Provident Mutual Life Insurance Company

The indispensability of the glucose tolerance test in the evaluation of glycosuria makes it well worth our while to subject this procedure to a critical appraisal from time to time. Although the amount of sugar in a given blood specimen can be very accurately determined, the estimation of glucose tolerance is a biologic procedure subject to many variables.

We have found the conventional one-dose test a satisfactory safeguard against unknowingly accepting diabetic individuals for insurance. The Exton-Rose test has also proven effective in weeding out the abnormals and has had the added advantage of being time saving.

However, we have become aware of a tendency for the Exton-Rose test to result abnormally in cases in whom a normal one-dose test was obtained. This discrepancy along with other inconsistencies of repeated glucose tolerance tests, in some individuals, has led us to study certain factors which influence the results of the glucose tolerance test.

We have seen insurance denied because emotional strain or faulty antecedent diet had produced an abnormal response to the glucose tolerance test, whereas later studies under more favorable circumstances resulted in a normal response. Tests carried out on capillary blood may wrongly be considered abnormal unless the widespread differences between

capillary and venous blood are taken into account. On the other hand, there are cases of supposedly benign glycosuria which have developed diabetes with the passage of time. Fortunately such occurrences are the exceptions; and from the majority of glucose tolerance tests valid conclusions may be drawn.

We shall consider five aspects of the glucose tolerance test. The subject matter of this discussion is based upon original work performed in our clinical laboratory supplemented by a review of the literature. Some of the original tables are omitted or abbreviated here but are available to anyone interested.

The micro-method of Folin-Malmros (1) was used throughout the following studies with the exception of one experiment. This has proven quite satisfactory in our hands as shown by numerous duplicate determinations.

THE NATURE OF "CAPILLARY" BLOOD SUGAR

Blood obtained by puncture of the finger is used frequently in insurance medicine. It is generally agreed that free flowing finger blood is arterial blood. However, some feel that squeezing the finger or using a cold hand results in a variable mixture consisting of arterial, venous, and capillary blood and even tissue juice or lymph. We feel it is important to establish the true nature of finger blood. Therefore we made a comparative study of the sugar content of blood obtained simultaneously from the radial artery, the arm vein, and multiple finger punctures during the hyperglycemia following glucose ingestion. The effect of various lancets and of squeezing and previous immersion in ice water were studied. We found that regardless of these variations in technique, finger blood sugar was practically identical with arterial blood sugar even in the presence of marked arterio–venous differences. For all practical purposes blood obtained from the finger (cutaneous or capillary blood) is synonymous with arterial blood.

DIFFERENCES IN ARTERIAL (CAPILLARY) AND VENOUS BLOOD SUGAR VALUES

Significant differences between arterial (capillary) and venous blood sugar values during the one-dose glucose tolerance test have been repeatedly demonstrated. Hagedorn (2), Foster (3), Rabinowitch (4), and others have found that marked differences may occur between arterial and venous blood values after the ingestion of glucose. Our experience with simultaneous arterio-venous curves after the administration of a single dose of 50 grams of glucose is summarized in Table I. Arterio-venous blood glucose differences during the glucose tolerance test may be of great magnitude especially during the peak.

In view of the fact that the two-dose, one-hour glucose tolerance test (Exton-Rose Procedure) (5) (6) involves a somewhat different physiological principle and presents the organism with an additional stimulus, it seemed worthwhile to ascertain the magnitude of the differences between arterial and venous blood glucose during this test. We were able to obtain 32 simultaneous arterial and venous blood sugar determinations during the course of glucose tolerance tests on presumably normal individuals. (7) These tests were made after a twelve hour fast—the blood was obtained from the finger and the vein simultaneously by two different operators. Almost all of the determinations were made in duplicate, that is two 0.1 c. c. quantities of blood were obtained from the finger and each was analyzed.

In the case of venous blood, the blood was placed in a test tube and before coagulation could occur, two 0.1 c. c. quantities of blood were withdrawn and each was analyzed.

We analyzed duplicate samples of blood to demonstrate that the arterio-venous differences which we obtained were not dependent upon any technical errors but were true differences in value.

TABLE I—TABLE II

TABLE I

ARTERIO-VENOUS DIFFERENCES IN BLOOD SUGAR DURING THE TWO-HOUR, ONE-DOSE TOLERANCE TEST

	FASTING	½ HOUR	2 HOUR
AVERAGE DIFFERENCE	0.9	17.8	7.3
MAXIMUM DIFFERENCE	14	35	30
MINIMUM DIFFERENCE	0	2	0

TABLE II

ARTERIO-VENOUS DIFFERENCES IN BLOOD SUGAR DURING THE ONE-HOUR, TWO-DOSE TOLERANCE TEST

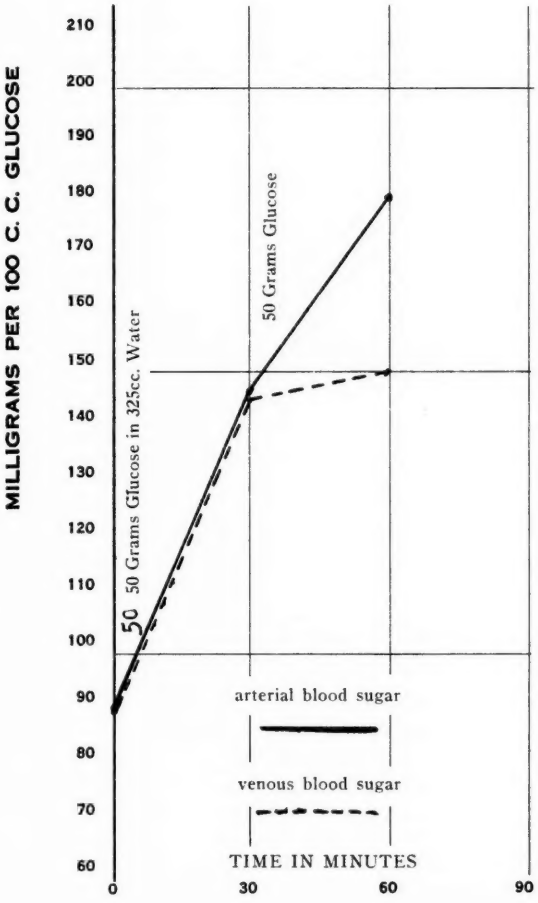
	FASTING	½ HOUR	1 HOUR
AVERAGE DIFFERENCE	1.8	13.8	21
MAXIMUM DIFFERENCE	11	33	57
MINIMUM DIFFERENCE	0	0	0

A summary of the results of the Exton-Rose tests is set forth in Table II. In general, the fasting blood glucose values are the same or nearly identical in both arterial and venous blood. A divergence at one-half hour is marked in some cases and absent in a few. At one hour the divergence is greater on the whole than at one-half hour and at times extreme.

One can not predict the arterio-venous difference from either the arterial blood sugar level or the venous blood sugar level

FIGURE I

ARTERIO-VENOUS DIFFERENCE
DURING THE
EXTON-ROSE PROCEDURE



alone. Exton–Rose curves on arterial blood may be especially misleading when the results are not within normal limits. Figure I illustrates this point.

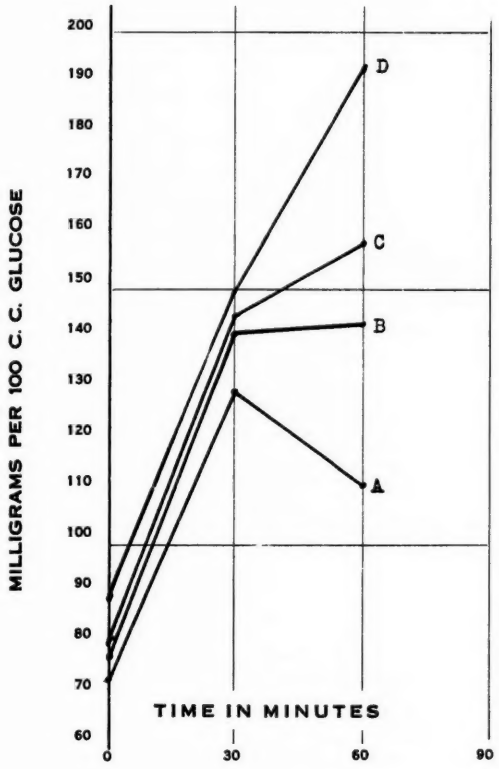
If the curve from arterial blood is normal, one may rest assured that the venous curve will likewise be normal, but if the arterial curve is questionable it is necessary to procure venous blood sugar values in order to obtain a true picture of the glucose tolerance.

It is the habit of some individuals to arrive at standards for interpreting arterial (capillary) blood by adding 10 or 20 mg.% to the values accepted as normal for venous blood. Our results show that the use of such an average conversion figure might well lead to error in the interpretation of certain individual curves, especially in the case of the Exton-Rose procedure.

THE INFLUENCE OF ANTECEDENT DIET UPON THE GLUCOSE TOLERANCE TEST

The influence of antecedent diet upon the conventional one-dose, two-hour glucose tolerance test has been adequately demonstrated, (8), (9), (10). Starvation, low-carbohydrate and high-fat diets cause a diminished ability to metabolize glucose in the normal individual with resultant abnormally high blood sugar levels following the ingestion of glucose. This situation is speedily corrected in the normal individual following a diet containing adequate carbohydrate. It has been stated that moderate variation of antecedent diet does not influence the Exton-Rose, two-dose, one-hour glucose tolerance test, (5) although Sweeney has shown that the Exton-Rose test is affected by extremely high-fat or high-protein diets in the absence of carbohydrate. (11)

We have found that certain individuals, especially those who know they have had glycosuria in the past, are apt to prepare for the test by taking a low carbohydrate diet for a



EFFECT OF ANTECEDENT DIET UPON
THE EXTON-ROSE, ONE-HOUR,
TWO-DOSE GLUCOSE TOLERANCE TEST

A. Average normal diet. B. During the preceding day, the diet consisted of: C-60 grams, P-70 grams, F-70 grams. C. During the preceding day, the diet consisted of: C-30 grams, P-60 grams, F-80 grams. D. During the preceding day, the diet consisted of: C-10 grams, P-80 grams and F-90 grams. (Am. J. Clin. Path. 11:43, 1941)

day or so beforehand. In some cases, a physician, finding glycosuria, has placed the individual on a low-carbohydrate diet. We have observed such instances in which the Exton-Rose glucose tolerance test was abnormal and subsequently was restored to normal by an adequate intake of carbohydrate.

We have obtained glucose tolerance tests by the Exton-Rose technique (5) in a number of experiments upon normal individuals before and after a period of special dieting and the results indicate that moderate as well as extreme manipulation of diet is capable of altering the Exton-Rose glucose tolerance from the normal to the diabetic type. (12)

We have selected Figure II to illustrate the effect of varying degrees of carbohydrate restriction in the same individual. In this experiment the antecedent diet was restricted for three meals and the tests were performed the following morning after a twelve hour fast. The lowest curve (A) is the normal curve for the individual on his average diet. Curve B shows the effect of moderate restriction in carbohydrates. Curve C follows marked carbohydrate restriction and curve D reveals the effect of a high-fat diet in the relative absence of carbohydrates. Venous blood was used throughout our study of diet.

COMMENT

These results illustrate the need for proper preparation prior to the Exton-Rose, as well as prior to other types of glucose tolerance tests. The antecedent diet should always contain a normal amount of carbohydrate. Undoubtedly some individuals with glycosuria due to a low renal threshold have been placed upon a low-carbohydrate diet and subsequently a glucose tolerance test taken while a state of impaired glucose metabolism existed. These cases are then suspected of being diabetic. A glucose tolerance test following proper antecedent diet may reveal normal blood sugar levels.

INSTANCES OF DISAGREEMENT IN THE RESULTS OF GLUCOSE TOLERANCE TESTS

In the course of performing 160 two-dose, one-hour glucose tolerance tests (Exton-Rose Procedure) it seemed that this test was more sensitive than the conventional one-dose test. We have selected instances where the one-dose, two-hour and the two-dose, one-hour glucose tolerance tests were performed on the same individual in order to determine the relative merits of the two tests and evaluate proposed criteria for their interpretation. (13)

A review of the literature reveals three papers dealing with a comparison of the two-dose and the one-dose tests.

Kelly, Beardwood and Fowler (14) found the two tests to be in close agreement and cite only two cases out of fifty where disagreement occurred when both tests had been performed in the same individual. These authors place their reliance in the two-dose, one-hour test. Young (15) studied four individuals with presumably normal carbohydrate metabolism and found the two-dose test to be abnormal in each instance whereas the conventional one-dose test resulted normally. He felt that the two-dose test was in error. Brown (16) reported the results of both tests performed in each of 24 subjects. He considered the two tests to be equally accurate.

METHODS

Individuals were selected upon whom we had obtained borderline or slightly abnormal two-dose tests, that is, a one-hour venous blood sugar value of 160 mg. per 100 c.c. or more by the analytical method of Folin and Malmros. At the time of study none of the group was overweight, arteriosclerotic, or suffering from an infection. As far as could be determined no other factor which might have influenced the glucose tolerance was operative. All of the individuals used in this study except D. A. and R. C. are well known to us and have had other studies of their glucose metabolism made.

TABLE III

COMPARISON OF BORDERLINE GLUCOSE TOLERANCE TESTS

SUBJECT		CONVENTIONAL ONE-DOSE, TWO-HOUR TECHNIQUE					EXTON-ROSE TWO-DOSE, ONE-HOUR TECHNIQUE				
		DATE	FASTING	½ HOUR	2 HOUR	GLYCOSURIA at 2 Hr.	DATE	FASTING	½ HOUR	1 HOUR	GLYCOSURIA at 1 Hr.
			mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent		mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent
1	L. D.	7-30-41	v† 80 c† 80	150 180	60 60	3.0%	7-3-41	v† 100 c† 95	163 190	180 230	4.0%
2	J. G.	5-27-41	v 112 c 112	180 200	102 107	1.5%	6-17-41	v 117 c 115	190 220	175 225	2.5%
3	J. S. P.	4-28-41	v 113 c 110	175 190	100 95	Neg.	4-24-41	v 106 c 104	152 154	192 222	Neg.
4	J. R.	8-1-41	v 85* c 95*	125 145	105 95	0.5%	7-30-41	v 98 c 93	135 142	160 190	No Report
5	E. B.	6-2-41	v 97 c 100	176 190	123 123	1.0%	6-23-41	v 90 c 90	170 173	180 190	2.0%
6	H. R.	8-11-41	v 102* c 97*	176 198	189 197	5.0%	6-16-41	v 92 c 95	160 165	166 178	1.0%
7	W. M.	8-13-41	v 88* c 90*	155 168	105 112	Neg.	8-27-41	v 88 c 88	175 185	190 200	0.5%

TABLE III Continued

SUBJECT		DATE	FASTING	½ HOUR	2 HOUR	GLYCO-SURIA at 2 Hr.	DATE	FASTING	½ HOUR	1 HOUR	GLYCO-SURIA at 1 Hr.
			mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent		mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent
8	H. L.	6-11-41	v 100 c 102	130 164	125 145	Neg.	8-25-41	v 95 c 98	152 165	175 182	Neg.
9	J. P.	8-28-41	v 90* c 107*	150 186	95 115	Neg.	9-4-41	v 85 c 90	150 155	160 181	Neg.
10	R. G.	11-6-41	v 98*	173	115	0.5%	10-30-41	v 82 c 85	155 174	175 210	0.3%
11	R. O.	10-16-36	v 85	175	100	No Report	4-2-35 6-25-36	c 125 c 92	225 148	235 160	0.3% 0.3%
12	K. B.	3-25-40	c 70	145	75	Neg.	1-16-40 6-17-40	c 85 c 97	185 160	185 180	0.3% 0.5%
13	D. A.	11-2-40	v 82* c 86*	128 192	81 114	Neg.	10-29-40	c 97	130	173	Neg.
14	R. C.	11-17-36	v 87*	118	72	Neg.	11-27-36	c 95	155	180	Neg.
15	A. B.	2-14-35	c 97	137	106	Neg.	2-7-35	c 112	145	187	Neg.
16	D. C.	5-30-40	v 132	156	176	No Report	5-22-40	c 110	150	175	2.0%

†v—Venous Blood Glucose Value c—Capillary Blood Glucose Value

*These subjects received 100 grams of Glucose in 325 cc. of water. All others received 50 grams in 200 cc. of water.

Three individuals were known to have had glycosuria with a normal glucose tolerance for a number of years. All of the subjects studied were in good health and without past history or symptoms of diabetes except in the case of H. J. R. and D. C. who were suspected of having latent diabetes. Care was taken to provide for an adequate antecedent diet.

These selected individuals were also subjected to the conventional one-dose, two-hour technique in which a single dose of either 50 or 100 grams of glucose was administered (Table III). Our criterion for a normal response to this test, using venous blood, requires that the fasting blood sugar shall be less than 120 mg. per 100 c. c., the one-half hour value shall be 180 or less, and the two-hour value shall be 120 mg. per 100 c. c. or less. These criteria are commonly used in insurance practice. Even if the critical two-hour level is reduced to 110 mg. per 100 c. c. it would not cause any difference in the interpretation of the results in Table III.

Both types of glucose tolerance tests were performed in the morning after an overnight fast. In ten of the cases we were able to obtain simultaneous samples of arterial blood from the finger and venous blood from the antecubital vein. In these cases duplicate determinations were performed on each sample of arterial and upon each sample of venous blood by the method of Folin and Malmros. The average of the duplicates in each instance is recorded in Table III. In the other six subjects only arterial or only venous blood was obtainable as will be seen upon consulting Table III.

Criteria for the interpretation of the two-dose, one-hour glucose tolerence test: According to the criteria of Exton and Rose (5) a normal response required (1) a normal fasting blood sugar, (2) a rise which did not exceed 75 mg. per 100 c. c. in the half-hour specimen and (3) an hour blood sugar value which did not exceed the half-hour value by more than 10 mg. per 100 c. c. We feel that their criteria were too strict in limiting the allowable rise during the second half-hour to 10 mg. per 100 c. c.

Gould, Altshuler, and Mellen (17) showed that a 30 mg. per 100 c. c. rise could occur during the second half-hour in a normal individual. Their other requirements were a normal fasting blood sugar level and a rise during the first half-hour not in excess of 50 mg. per 100 c. c. These authors concluded that if at least two of the three requirements were fufilled the individual could be considered as normal.

Matthews, Magath, and Berkson (6) found that the one-hour blood sugar value served as the most useful criterion and they did not depend upon the rise or fall pattern of the curve. Their critical level was established as 158 mg. per 100 c. c. at the end of one hour when samples of venous blood were obtained and analyzed by the method of Folin and Wu. Below this level none of the individuals studied were diabetic. When the hour blood sugar level was between 160 and 180 mg. per 100 c. c. the case was considered borderline since blood sugar values in 5.1% of normals and 7.7% of very mild diabetics studied by these authors fell into this range. When the one-hour value was in excess of 180 mg. per 100 c. c. all the individuals observed proved to be diabetic.

We accepted the critical one hour level of Matthews et al. because it diagnosed all of our cases of diabetes correctly, whereas the criteria of Gould et al. failed to make the correct diagnosis of diabetes in four cases observed by us. The highest one hour value in our series obtained in the case of a presumably normal individual was 192 mg. per 100 c. c. of venous blood.

Let us confine our attention to the venous blood sugar values for the two-dose test in the first ten cases. According to the criterion of Matthews et al. two subjects would be considered diabetic, eight borderline, and none normal. According to the criteria of Gould et al. all ten individuals would be considered normal. According to the one-dose test seven subjects would be considered normal, two are borderline, and only one is diabetic. Clinical data substantiates this diagnosis of diabetes in case #6 (H. R.).

In the last six cases of Table III only arterial blood sugar values are available for the interpretation of the two-dose test. From our experience with arterial blood we would consider the first two-dose test performed upon R. O. to be diabetic whereas the later test is normal. The tests in cases 12 to 16 are all in the borderline range. According to the one-dose test, cases 11, 12, 13, 14, and 15 are clearly normal where case 16 is diabetic. Case 15 (A. J. B.) first observed in 1935 (Table III) is now a diabetic requiring insulin. Case 16 (D. C.) since his borderline two-dose test in 1940 (Table III) has been considered to be a mild diabetic.

To summarize the results we may say that in 3 out of 16 cases a borderline two-dose test has accurately diagnosed diabetes; in two of these instances the one-dose test was in agreement. In 13 cases an abnormal or borderline two-dose test has occurred in presumably normal individuals; in 2 of these instances the one-dose test was also either top normal or slightly borderline.

COMMENTS

The results presented indicate that the two-dose glucose tolerance test of Exton and Rose is usually more sensitive than the one-dose test. We may draw one of two conclusions: either the two-dose test is more accurate than the one-dose test in detecting potential diabetes or the two-dose test is over-sensitive and may be used as an exclusion test but in borderline cases should be rechecked by performing a conventional one-dose test.

At this point we are inclined to believe that the two-dose test of Exton and Rose possesses greater sensitivity but that the conventional one-dose test is more specific for diabetes.

We have accepted the criterion of Matthews et al. in interpreting our studies on the two-dose test because it did not fail to diagnose a single case of diabetes in our experience. Their criterion is more sensitive but less specific than the criteria of Gould et al. We think it is wise to err on the side

of safety and accept the more sensitive criterion. In accordance with this point of view, any individual with an hour blood sugar value in excess of 160 mg. per 100 c.c. by the analytical method of Folin and Malmros or by the method of Folin and Wu should be suspected of having diabetes until proven otherwise. It is apparent from our study that there is a borderline range between 160 and 190 mg. per 100 c.c. where the results may indicate either normalcy or diabetes; in this range prolonged observation may be necessary.

It is obvious from the results of the above discussion that the incidence of disagreement between the two-dose and the one-dose tests will depend partly upon the criteria used in interpreting these tests. Had we adopted the criteria of Gould et al. we should have considered the one-dose test more sensitive than the two-dose test.

SUMMARY

From our group of 160 subjects who had submitted to the two-dose, one-hour glucose tolerance test, 16 cases were studied in which the results were borderline or slightly abnormal. A one-dose test was also performed on these individuals. In 13 instances the one-dose test and the available clinical data indicated a normal carbohydrate metabolism and therefore were in disagreement with the two-dose glucose tolerance test. In view of the better established criteria for the conventional one-dose glucose tolerance test, we believe that it lends itself more readily to underwriting and results in fewer unwarranted declinations than the newer Exton-Rose procedure.

NON–DIABETIC GLYCOSURIA

It has been repeatedly stated that non-diabetic glycosuria is without serious consequences. Marble, Joslin, Dublin, and Marks (18) state that "Non-diabetic glycosuria in itself has little or no adverse influence upon longevity." These authors point out the paramount importance of adequate initial study

to establish the true nature of the glycosuria. John (19) is of the opinion that "There is sufficient evidence in the literature to support the statement that non-diabetic glycosuria remains so throughout the years.....and is not a transitory state toward diabetes." Lawrence (20) writes as follows: "If normoglycemic glycosuria is proved by a thorough investigation, such as the finding of a normal glucose tolerance test, then I think the whole condition can be neglected at once and forever." In general, our studies tend to substantiate these and other statements in the literature. (21)

METHODS:—Subjects were chosen from a group of individuals who had submitted to glucose tolerance tests 5 to 13 years ago because of a previous finding of glycosuria in the course of an examination for life insurance. Most of the subjects selected for follow-up had clearly normoglycemic curves with glycosuria, but a few borderline glucose tolerance curves are included for comparison. One hundred and nine individuals were invited to participate but only 35 accepted. Five additional cases could be followed up from existing records. The majority of cases were restudied with the conventional one-dose, two-hour test employing one dose of 50 grams of glucose. A few cases recorded in the latter portion of Table IV were studied by the Exton-Rose, two-dose technique (5) in both the original study and the follow-up or they may have received the one-dose test in one instance and the two-dose in another. The figures reported in Table IV represent arterial (capillary) blood sugar values.

RESULTS:—Of the 40 cases that were restudied after a period of 5 to 13 years, 4 developed abnormal glucose tolerance curves. In one of these (case No. 18) the original study had resulted in a borderline blood sugar curve although a two-hour value of 146 mg. per 100 c.c. of arterial blood may be seen in a non-diabetic individual. In the other three cases (cases Nos. 10, 34 and 37) the blood sugar figures were definitely within normal limits at the time of the original examination although one case (No. 10) had a two-hour value at

the upper limit of normal and another case (No. 34) had a one-half hour value that was somewhat high. On the other hand, many of the individuals who did not develop diabetes had original blood sugar curves as high or higher than those individuals whose blood sugar tolerance curves were abnormal on follow-up study. (Table IV)

The percentage of sugar excreted in the urine seemed to be of no prognostic value, indeed, curiously enough, in the individuals we studied those who excreted the most sugar did not develop diabetes. (See Table IV)

DISCUSSION:—As early as 1922 Holst (22) found the causes of non-diabetic glycosuria to be an abnormally low renal threshold, high alimentary rise, or a combination of the two. Marble and Joslin's (18) definition of true renal glycosuria requires the presence of glucose in fasting as well as post-prandial urine, an unusual condition. The great majority of non-diabetic glycosurias have a threshold that is exceeded only after the ingestion of a moderate amount of carbohydrates. None of our subjects would qualify as renal glycosuria according to Joslin's definition. With regard to intermittent and continuous glycosuria Fischer (23) feels that it seems unnecessary to separate arbitrarily the two types of renal glycosuria for they are benign, frequently familial and are differentiated only by the height of the renal threshold for glucose. Regardless of one's classification of non-diabetic glycosuria it is the consensus of opinion that the condition is benign. John (19) in a summary of the literature has shown that individuals may put out large quantities of sugar for years without detriment to themselves. Our results are in agreement with this finding. (Table IV) Thus it is evident that the excretion of a large amount of sugar is not necessarily associated with an unfavorable prognosis. When one obtains an unequivocally normal blood sugar tolerance curve especially on more than one occasion it seems unimportant whether the individual has little or much glycosuria. The amount of glycosuria with normal post-absorptive blood sugar levels is purely

TABLE IV

FOLLOW-UP STUDY OF NON-DIABETIC GLYCOSURIA

One-Dose, Two-Hour Glucose Tolerance Test Used Except in Those Cases Marked with an Asterisk.

*Indicates a One-Hour Value of the Two-Dose, One-Hour Technique (Exton-Rose).

	ORIGINAL STUDY						FOLLOW-UP STUDY				
	SUBJECT	DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA	DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA
			FASTING	½ HOUR	2 HOUR			FASTING	½ HOUR	2 HOUR	
			mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent		mg. per 100 cc.	mg. per 100 cc.	mg. per 100 cc.	percent
1	E. P. W.	7-13-31	113	190	125	0.5%	2-19-37	105	200	66	+
2	L. R.	8-15-34	65	130	80	3.0%	8-25-39	120	140	95	1.0%
3	G. M.	3-10-34	106	171	88	+	3-17-40	85	175	92	0.4%
4	R. S. M.	3-9-37	88	157	80	3.0%	12-30-40	76	—	93	2.0%
5	J. P.	2-26-29	110	206	70	+	4-28-41	110	190	95	0.5%
6	E. D. B.	12-10-29	114	180	96	0.5%	6-2-41	100	190	123	1.0%
7	J. W. W.	1-28-30	105	206	71	+	5-14-41	78	143	90	1.0%
8	E. L. S.	9-25-30	100	196	127	+	7-19-41	75	112	130	0.5%
9	G. S. M.	7-14-31	109	171	147	0.4%	6-3-41	90	133	83	0.3%
10	W. H. B.	1-26-32	99	178	118	1.0%	5-13-41	110	168	185	10.0%
11	J. H. M.	4-7-32	91	167	121	0.2%	6-25-41	82	162	110	0.5%

TABLE IV Continued

ORIGINAL STUDY							FOLLOW-UP STUDY				
SUBJECT		DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA	DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA
			FASTING mg. per 100 cc.	½ HOUR mg. per 100 cc.	2 HOUR mg. per 100 cc.	percent		FASTING mg. per 100 cc.	½ HOUR mg. per 100 cc.	2 HOUR mg. per 100 cc.	percent
12	H. S.	7-5-32	92	155	96	0.5%	6-11-41	80	120	70	0.2%
13	J. P. G.	10-19-32	110	158	145	1.0%	5-27-41	110	198	105	1.5%
14	W. B.	2-9-33	97	123	133	0.4%	6-25-41	90	115	83	0.0
15	D. H. W.	3-31-33	95	121	101	1.0%	6-18-41	90	132	97	0.5%
16	O. W.	8-8-33	93	161	86	0.5%	5-10-41	75	102	60	0.3%
17	J. M.	9-22-33	91	182	108	1.0%	6-18-41	92	103	73	0.0
18	M. M.	11-17-33	98	167	146	0.2%	6-26-41	120	230	185	3.0%
19	M. S.	3-27-33	—	153	74	0.4%	5-27-41	88	138	94	0.5%
20	H. W. M.	12-13-33	112	270	132	1.0%	7-6-41	85	183	93	0.2%
21	A. R.	7-31-34	109	155	88	0.4%	5-15-41	88	134	82	0.5%
22	E. H. E.	12-2-35	80	174	100	0.5%	6-22-41	96	135	92	0.5%
23	F. H. O.	12-9-33	110	161	95	1.0%	8-16-41	90	136	97	3.0%
24	G. H. H.	5-31-33	80	180	99	1.0%	8-16-41	95	176	105	5.0%
25	S. C. H.	6-20-31	114	168	157	0.3%	8-14-41	83	115	121	0.0
26	J. M.	12-11-34	80	125	85	1.0%	1-17-42	75	140	70	4.0%

TABLE IV Continued

ORIGINAL STUDY							FOLLOW-UP STUDY				
SUBJECT		DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA	DATE	BLOOD SUGAR TOLERANCE			GLYCO-SURIA
			FASTING mg. per 100 cc.	½ HOUR mg. per 100 cc.	2 HOUR mg. per 100 cc.	percent		FASTING mg. per 100 cc.	½ HOUR mg. per 100 cc.	2 HOUR mg. per 100 cc.	percent
27	T. J. B.	6-29-30	90	204	95	+	1-6-42	95	145	80	0.0
28	A. H.	2-27-37	80	138	80	+	2-25-42	100	145	80	0.5%
29	D. F. B.	9-21-29	80	145	80	3.0%	Known Glycosuria since 1924 Recent Fasting Blood Sugar 84 mgs.				
		1-7-32	83	208	83	2.0%					
30	R. O.	6-25-36	92	148	160*	0.3%	7-29-41	100	195	155*	0.5%
31	J. V. L.	3-3-37	80	150	180*	4.0%	8-5-41	78	130	135*	0.3%
32	F. C. B.	8-3-36	88	142	152*	1.5%	3-19-41	85	199	173*	0.5%
33	W. H. D.	6-18-36	90	150	146*	0.5%	8-12-41	90	140	154*	0.4%
34	E. S.	12-26-34	100	210	105	0.3%	8-1-41	95	203	273*	3.0%
35	L. C. S.	12-15-34	97	157	110	0.5%	5-8-41	94	150	140*	0.5%
36	L. D.	12-17-35	100	160	180*	1.0%	7-30-41	80	180	60	0.5%
37	H. J. R.	5-22-36	95	150	155*	1.0%	8-11-41	97	195	200	5.0%
38	H. H. M.	1938	Known Glycosuria since 1932				4-13-42	90	140	135*	0.3%
			95	125	115						
39	J. R.	10-11-32	88	125	124	1.0%	3-17-42	88	105	140*	0.5%
40	O. O.	12-12-35	80	165	168*	1.0%	4-18-42	94	140	50	0.2%

and simply a function of the renal threshold for glucose. In discussing non-diabetic glycosuria, Marble, Joslin, Dublin, and Marks (18) state:—"Caution must be advised against accepting as normal, tolerance curves which either at the highest point or subsequent fall are on the borderline of the type found in mild diabetes." We believe that the importance of this cannot be over-emphasized and in our experience this statement is equally true whether there is glycosuria or the urine is sugar free. Any borderline glucose tolerance curve should be rechecked by a repetition of the test after all the factors which may have influenced it have been considered and corrected.

Of the 109 cases of glycosuria with normal or borderline glucose tolerance tests which we attempted to follow up, 8 could not be contacted because of incorrect addresses, 19 did not reply to two or more letters, 32 either declined to have the study done or accepted but failed to keep their appointment for the test; of these at least 3 are known to have developed diabetes, 10 of the individuals had died, but in none of these was diabetes mentioned as a contributing cause of death, and 40 yielded the information which is summarized in Table IV. All the individuals in the group studied were symptom-free and considered themselves in good health, even those with apparent diabetes.

An interesting question presents itself. Why did 3 individuals with normal blood sugar curves out of a group of 40 cases of glycosuria develop probable diabetes? This is much higher than the average chance of developing diabetes. Several possibilities suggest themselves:—(1) Obviously a technical error in the original study may have occurred in the determination of the blood sugar curve. (2) The reason for performing the original study was the previous finding of glycosuria; this may have been due to a diminished ability to metabolize glucose. Repeated glucose tolerance curves upon the same individual will show considerable variation. The original glucose tolerance curves in our study may have been

performed at a time when a coincidence of favorable circumstances produced a tolerance curve within top normal limits in a very mild diabetic with a lower than average renal threshold for glucose. This possibility emphasizes the need for more than one tolerance study in the evaluation of this type of case. (3) High peak values which some observers consider to be of prognostic significance may have been missed by taking specimens at only one-half and two hours. (4) Our study tends to disprove the possibility that the diabetic organism has more tendency to excrete glucose than a normal individual at a given blood sugar level. In fact, this possibility seems very unlikely since both mild diabetics with only moderately elevated blood sugar levels and severe diabetics with high blood sugar levels may fail to excrete glucose due to a high renal threshold for glucose.

Whatever the reason, it seems to be a fact that when conclusions are drawn from a single glucose tolerance test, an individual with apparently non-diabetic glycosuria is more likely to develop diabetes than a normal individual. This has also been the experience of Marble et al. (18).

SUMMARY

1—In a group of individuals with apparently non-diabetic glycosuria based upon one glucose tolerance curve obtained 5 to 13 years ago a greater number developed diabetes than can be accounted for on the basis of chance.

2—The amount of glucose in the urine was, in itself, of no prognostic significance.

3—If glycosuria is proven by thorough study and continued observation to be truly non-diabetic, it is without detriment to health.

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PRESIDENT WENSTRAND—We are very fortunate that Dr. William G. Exton has agreed to discuss this paper of Dr. Dewees. Dr. Exton has spoken to us a good many times before and needs no introduction to this audience. Dr. Exton!

DR. EXTON—First, let me compliment the essayists on the good work they have done, because it is impossible to do too much of it in bringing out the truth. Let me also say that until Dr. Rose and I gave the two-hour test at our 1931 Hartford meeting, the conventional test was 100 grams of glucose observed over three hours. Dr. Rose and I have always considered the test, which the paper now calls conventional, as just as much our baby as the one-hour test and it sort of puts me on the spot to talk about them.

The essayists cover the subject very fully, and I shall, therefore, briefly discuss only three points:—the differences between arterial and venous blood sugar,—the effects of antecedent diets,—and the criteria of the one-hour test.

The essayists prefer venous over arterial blood sugar values and their choice is quite natural because all of us are more familiar with the older and richer data on venous blood than we are with the relatively recent and scant data on arterial blood. We used finger prick blood in our pioneer work instead of venous, only because our Medical Directors would not risk veni-punctures by the examiners and their decision delayed work on the test until we devised a satisfactory method for determining blood sugar in O.1 c. c. samples.

I am sorry the paper does not have an illustration of the one-dose test for comparison with its illustration of a two-dose test, because the greater difference between simultaneous arterial and venous blood sugars I have ever seen was reported by Doctors Joslin and Root on a one-dose test. The truth is, of course, that these arterio-venous differences are common to all oral sugar tolerance tests.

In the past year we have gained some insight into these differences through our work on blood oxygen. There are many similarities in the way the body utilizes sugar and oxygen, although it can store an abundance of sugar, but needs a continuous replacement of its oxygen supply. When arterio-venous differences exist, I call your attention to the fact that the venous values of both sugar and oxygen are always lower than the arterial. I call your attention also to the fact, that the venous values of both sugar and oxygen differ in samples of blood from different veins of the same individual, while the arterial values of both sugar and oxygen are always the same in blood samples from different arteries of the same individual. This is well illustrated by a recent study of arterio-venous differences made by Gibbs and others (Jour. Biol. Chem.–Vol. 144, 1942).

They measured the amounts of sugar and oxygen consumed in the brain by subtracting the amount in blood from one specific vein;—the internal jugular, from the amount in blood from either brachial, radial and femoral artery. In other words, they could count on getting the same answer from any artery, but not from any vein, because blood samples from different arteries give the same answer but samples from different veins may not.

From what I can gather, the physiologists seem to prefer arterial blood samples. Thus, in the chapter on glucose clearance in his "Physiology of the Kidney" Homer Swift states that considerable differences may exist between the concentrations in arterial and venous blood and the use of venous blood may lead to a deceptive value, etc.

If you take simultaneous blood samples and plot the results of one and two-dose tests on top of one another, you will find that the terminal blood sugar of the one-dose test is higher than that of the two-dose test, especially in borderline and diabetic cases. You will also find that the difference between arterial and venous blood is greater, because there is a definite relation between the spread of the arterio-venous

difference and the amount of sugar. The more sugar the more spread.

From all these considerations, one gathers that arterio-venous blood sugar differences are inherent in all oral sugar tolerance tests; and that a preference for arterial rather than venous blood sugar values may be more correct.

In the matter of antecedent diets, I am again sorry that the paper fixes attention on only one of the tests. Fortunately, excellent data on both tests have been supplied by others.

Some years before we published the two-dose test, Dr. Sweeney studied the effects of starvation and exclusive protein, fat and carbohydrate diets by feeding them to a group of healthy young adults forty-eight hours before making one-dose tests. (Sweeney, J. S.–Archiv. Int. Med. Dec., 1927.) He found that an exclusive carbohydrate diet gave lower sugar values and a more normal curve than starvation and the other exclusive diets, which gave higher sugar values and more abnormal curves. After publication of the two-dose test, Dr. Sweeney repeated his feeding experiments in exactly the same way before making two-dose tests (Sweeney, J. S.–Jour. Clin. Path. Nov., 1937) and with about the same results, although he states that the diets affect two-dose tests somewhat less than one-dose tests when there are differences.

As a practical matter for you gentlemen engaged in acting on risks, there is nothing in all this diet stuff for you to worry about, no matter what test you use. If you order a tolerance test because there is a suspicion of diabetes and an applicant grooms himself for the test by starving or excluding carbohydrates from his diet in the mistaken notion it will help him to get through you, nature has given you a break, which I think you need not be too sad about.

In other kinds of diet cases, like those who say they have not fasted overnight and must be tested an hour or so after

eating this or that for breakfast or lunch, a one-hour two-dose test is much easier to interpret than a two or three-hour test.

One of the consequences of publishing the one-hour test has been a very considerable correspondence from doctors who send me curves to interpret, or who favor me with their data, or who ask about criteria, especially about changes and I am glad to take this opportunity to tell you something about changes in the criteria.

When work on the test began, my clinical connections had been severed several years and I did not have proper clinical material to work with. I used clerks who worked every day in our department for normals, a few cases found in the office of our Longevity Service for borderline and mild diabetics and sponged on my medical friends for blood samples from some of their diabetic patients. I had no control of even this inadequate material and was so keenly conscious of its deficiencies for building a new test, that I laid the data aside after assembling it and deriving its criteria.

Later on, when a clinical group invited me to read a paper at a meeting in Cleveland and I had nothing else new at the time, I gave them just what we had with the best criteria we could find in our poor material. Although I was much surprised at the great amount of interest shown by the lively discussion of the paper and the many questions put to me, I told them quite freely about the defective nature of the material on which the criteria were based and expressed the hope that some one who had control of an adequate clinical material would study the test and perhaps improve the criteria. That, I am glad to say, has happened.

Over thirty papers dealing with the test have since then been brought to my attention, but I shall refer now only to the one by Dr. Wilder's associates at the Mayo Clinic, because their study of 491 subjects, aged 2½ years to 75 years, was made under ideal conditions (Matthews, Magath and Berkson–Jour. A. M. A., Oct. 21, 1939).

Before testing 117 normals, 70 renal glycosuries, 247 latent, mild or borderline diabetics, and 57 more severe diabetics, each subject had been studied, diagnosed and graded according to severity by Dr. Wilder.

Their results show clearly that no person with a blood sugar under 155 mgs. at the end of the test hour is diabetic, and that all persons with a blood sugar over 179 at the end of the test hour are diabetic. I am glad to say that the checks I have been able to make corroborate that conclusion and also their suggestion that the shape of the curve may be ignored when blood sugar at the end of the test is less than 155 mgs. Some disturbance of carbohydrate metabolism other than diabetes is indicated when the curves of such cases are diabetic in type. Furthermore, it is quite all right, as the Mayo Clinic authors suggest, to make the test with a single blood sample, when one is interested only in the diagnosis of diabetes.

On the other hand, in the small group of doubtful cases having blood sugars between 155 and 180 mgs., I think you will find that the shape of the curve will tell you if a case is diabetic or not.

Let me emphasize a warning. The Folin-Wu macro method was employed to measure blood sugar in the Mayo Clinic study. Blood sugar methods differ in the results they give. The criteria which have been given therefore, hold true only when the Folin-Wu macro method, or some other method which is known to give the same results, is used to measure blood sugar. We use the Folin-Wu method modified for 0.1 c. c. blood samples. This was developed in our laboratory and gives the same results as the macro method. We have never published it, but shall be glad to give details to anyone who is interested.

In conclusion, I may say that all of the evidence I have ever seen, including Dr. Dewees', shows that the one-hour two-

dose test is more specific and reliable than other alimentary glucose tolerance tests.

PRESIDENT WENSTRAND—Dr. Langner, have you anything else to bring out or to say?

DR. LANGNER—We want to thank Dr. Exton for his discussion.

The original test that we used was not the three-hour test but the one-dose two-hour test, using 50 grams of glucose.

Dr. Folin suggested using 50 grams of glucose in the taking of samples of blood at fasting, one half-hour and two hours, and that has been our procedure since. In 1931, Dr. Exton suggested 75 grams of glucose be used.

About the greatest difference reported between capillary and venous blood is 80 milligrams per cent, originally reported by Foster and later by Joslin.

With regard to antecedent diet, it would appear that if an applicant prepares himself by an antecedent diet he is trying to cheat a little bit. Some people feel that in order to prepare themselves for a glucose tolerance test that it is necessary for them to abstain from eating sugar, and in most instances an abnormal curve was obtained. When we told these people that the proper diet should be just the opposite, that they should take a diet high in carbohydrates, they proved to be perfectly good risks.

PRESIDENT WENSTRAND—Gentlemen, we shall have to change the order of the program a bit, inasmuch as Dr. Horrax has not arrived. Trains are usually late at this time of year, and especially during this emergency. Therefore, I am going to ask Dr. John L. Humphreys, in Dr. Eakins' absence, to read his paper. Dr. Eakins is laid up, not seriously, and he will be back again one of these days, but Dr. Humphreys did not have an opportunity to see this paper until just a little while before lunch, so he asked me to mention that fact in case he

stumbles over a word here or there. As you know, Dr. Eakins is very apt to use unusual words. Dr. Humphreys!

DR. HUMPHREYS—It was with considerable regret that Dr. Eakins was forced to forego this 1942 meeting due to illness, especially when he had been placed on the program. To all, he sends greetings for a successful and all-around enjoyable meeting.

This paper is short but succinct, and the more I have read it, the more the meat which is in it becomes apparent. The matters spoken of occur too often for enjoyment by those in our medical departments. May I say that I am sorry Dr. Eakins could not read this paper and bring it to you in his own inimitable manner.

BREAKING THE RULE OF THUMB

By O. M. Eakins, M. D.
VICE PRESIDENT AND SECRETARY
Reliance Life Insurance Company of Pittsburgh

All will agree that the pollex is a most necessary digital member. Those who delve into anthropology give us the interesting point that control and use of the thumb contributes to the fact that man is a greater, rather than a lesser, animal. Man's imagination has added to the stature of this advantage with simple and unmistakable symbolism.

If all the lack of exercise the thumb has achieved for hitchhikers were laid end to end, as our Actuarial friends are wont to do, it might aid in the study of muscular degeneration. The narial gesture with the thumb is certainly a grobianism, but none can gainsay its acidity of expression. Currently, the vertical thumb mutely "speaks volumes" in maintaining favorable psychology during the misfortunes of war.

In spite of the generations in which the thumb has been used popularly as a unit of measure, and the centuries in which it has denoted popular approval or disapproval, the "Rule of Thumb" is a standard with all the dithering qualities we associate with jelly-fish. And yet, it still seems to be dusted off, now and again, as a convenient weapon with which to confront Medical Directors.

"Simon says thumbs up. Simon says thumbs down. Simon says wiggle-waggle."

According to that traditional child's play, one errs in making a motion unless "Simon says." And although we, presumably, are not habromaniacs nor engaged in child's play, we do meet with "thumbs up" or "thumbs down" attitudes. And when "Simon says," some of us "wiggle" and some of us "waggle"—for the "Simon" in our case is that great American institution, the Life Underwriter.

Exceptions may prove the rule, in some instances—but not in life insurance medical underwriting. In our job of coldly evaluating risks with the aid of experience tables and medical facts, exceptions are apt to prove embarrassing. Especially so if they are made in isolated cases under a barrage of fast talking on the part of interested agents who make their livings by selling ideas.

Far be it from us to keep any mortal from making a living. It is so much easier to comply rather than deny. It is so much easier to let a majority have its way by the "Rule of Thumb" rather than stick to the letter of scientific principles which represent the minority report.

The entire system of life insurance protection, however, is based upon judicious selection along proven paths. Medical Directors, fortunately or unfortunately, as you wish, are sentries of the system. If we permit anyone without proper credentials to enter the camp of those who rest upon the assurance of our protection, we place the entire system in jeopardy and are unfaithful to the confidence and trust placed in us.

In the unusual circumstances rife in the world today, it is expected that some of the rules upon which we have based decisions in the past will be found too elastic and will have to be tightened. Others will be too stringent and must be loosened. These revisions will be necessary to meet fundamentals of medical underwriting.

Revision of rules to apply to everyone, however, is something else again compared with bowing to the "Rule of Thumb" under pressure to show leniency to a few. To make a habit of convenience to meet necessity results only in expediency. The right thing, and the expedient thing may be quite different. Real issues should be faced and solved. If rightly solved, then success attends the solution; conversely, failure. Perfunctory and casual disposal to satisfy an individual or a few in all probabilities will prove eminently unsatisfactory to all in the end.

Preparedness to anticipate necessary action, and everlasting determination to stand by our guns and stick to decisions known to be accurate, regardless of all influences to the contrary is the cure for the "Rule of Thumb." Any other course is merely a palliative, but the headache will remain.

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PRESIDENT WENSTRAND—Some of you probably remember that a few years ago, Dr. Eakins dilated very much upon the medical director. I reread some of that a little while back and this paper reminds me of his way of bringing out serious things in a lighter vein.

The next speaker on our program is Dr. Gilbert Horrax. Dr. Horrax, associated with The Lahey Clinic in Boston as neurological surgeon, was graduated from Johns Hopkins but then transferred his allegiance to Harvard, serving during his years of training at Peter Bent Brigham Hospital and as Arthur Tracy Cabot Fellow in charge of Laboratory of Surgical Research, Harvard. After a year as Resident Surgeon at the Massachusetts General Hospital, he received a commission as Major, Medical Corps, U. S. Army, serving from 1917 to 1919 in the first World War. In addition to many other positions of trust, he was both Associate and Senior Associate in Neurological Surgery at Peter Bent Brigham from 1919 to 1932, working with Dr. Harvey Cushing. Dr. Horrax is a member of many distinguished societies. He was Vice President of the American Neurological Association in 1941, and President of the Society of Neurological Surgery 1937–1938. Dr. Horrax, I am very glad to present you to this body.

CRITERIA OF PROGNOSIS AFTER HEAD INJURIES AS TO LONGEVITY AND DISABILITY

By Gilbert Horrax, M. D.‡
BOSTON

As medical directors of insurance companies of the United States and Canada, you are fully aware of the difficulties and complexities of the problem about which I have undertaken to speak to you this afternoon. Its ramifications are extreme, and certain aspects spread out from the physician and the hospital staff, by whom the patient is first treated, to the whole fabric of our industrial, legal and social activities. In many instances specific data from which conclusions might be drawn are entirely lacking, and in many others, judgment is difficult even in the presence of accurate records. What I shall endeavor to do is to give you some sort of a working basis which may be of assistance in evaluating an individual case, but in the last analysis the clinical judgment of experienced persons like yourselves, aided by the opinions of specialists or by the results of any specific tests which seem indicated, will form the final basis for such action as is deemed wise.

1. *Historical data.*—Much of value may be learned from an accurate account of the injury itself. This should include, when available, the exact nature of the accident, with particular reference to loss of consciousness and its duration, together with the duration of posttraumatic amnesia. From the latter one can estimate roughly the period of time necessary for rehabilitation** (Cairns) (1), although, as emphasized by Symonds (2), there are many drawbacks and limitations to even a roughly predicated schedule. More important, of course,

‡From the Department of Neurosurgery, The Lahey Clinic.

**The periods of time given by Cairns, even though rough estimates, are somewhat too specific to be taken as a true guide, and furthermore many other factors may need careful consideration after the patient has recovered from his amnesia.

is the type of injury, whether a skull fracture occurred and, if so, whether it was linear, depressed or compound, either at its site or indirectly through the ear drum or the paranasal sinuses. In view of judgment to be passed on persons involved in the present war the question of retained foreign bodies of any kind is particularly pertinent.

One should also know as much as possible concerning the treatment the patient received both at the time of the injury and subsequently in a hospital or at home. Specific data as to the length of time treatment was necessary and of what it consisted are of value. The results of examinations by a neurologist, psychiatrist or other specialists are pertinent, together with such information as may have been obtained by lumbar puncture, roentgenologic examination, electro-encephalogram and air encephalograms, if these were performed. Were there visual defects of any type, and if so, were they corrected or capable of correction? Also important, in those patients who need such advice, is the time interval after the accident before neurologic or other specialized neurosurgical advice was sought. This may have considerable to do with subsequent disability. According to Jefferson (3), this interval was anywhere from three weeks to two years, and he says specifically, "Many were not seen until seven, eight or nine months or more after injury. By that time a neurosis had become firmly established in a number of cases, and few were within measurable distance of returning to their work."

An extremely important factor in the history of any head injury is the presence or absence of local sepsis. This can be inferred from the length of time required for the healing of a scalp wound, or there may have been actual evidence of sepsis particularly in injuries compounded with the paranasal sinuses of the external ear.

Finally, the family history, together with the patient's own background may play an important role as to his future, the concern being centered largely upon emotional factors.

2. *Examination of the patient.*—In addition to a thorough

physical examination, the patient should have a careful neurologic and at times a psychiatric study. To these should be added the opinions of a psychologist and a psychometrist, if the situation warrants more detailed information. These specialists should submit their reports as consultants, and their findings, together with all the other evidence, should be taken into careful consideration in passing final judgment on the case.

Routine roentgenologic examinations of the skull should, of course, be taken in every case of head injury no matter how trivial it seems. Sometimes linear or depressed fractures are disclosed unexpectedly, and in the case of war injuries there may even be retained foreign bodies, such as bomb splinters or small bits of shell which had penetrated the skull through an apparently insignificant scalp wound.

Repeated electro-encephalographic studies are a recent and valuable means of determining the progress toward a normal state in patients who have suffered cranio-cerebral injuries (Williams) (4). Shortly after the injury and for a variable time thereafter, abnormal brain waves are present, and these tend to become more and more normal as time passes. The demonstration objectively of this abnormal brain activity may well turn out to be one of the best methods of gauging the disability of a patient, and may be of particular value in those patients suspected of having a "compensation neurosis," but who, in reality, have more brain damage than can be demonstrated by the older methods. In like manner the opposite may be found, that is, normal waves in a person who was thought to have organic difficulty. As yet, however, the result of electro-encephalographic examinations cannot be taken as absolute, but must form another link in the chain of evidence governing our judgment in an individual patient.*

* It may well be that the greatest use of the electro-encephalogram in neurologic or neurosurgical diagnosis will be found in the part it plays in determining the return to normal in postconcussion states. Much work on this problem is in progress in England and likewise in in the United States.

The use of air or oxygen encephalography undoubtedly is indicated in a small but perhaps increasing group of cases. This procedure is carried out almost as frequently as lumbar puncture in all neurosurgical clinics, and if skillfully performed under intravenous pentothal anesthesia, entails no discomfort. A varying degree of headache follows the procedure, but this rarely lasts longer than twenty-four hours and is largely relieved by codeine and aspirin. The risk is so negligible as to be practically nonexistent.

Air encephalograms are especially indicated for patients who may be suspected of harboring a chronic subdural hematoma or a quiescent brain abscess. Such a study may also show decided abnormality of the subarachnoid spaces, a cortical scar or a traumatic porencephalic cyst. In rare instances, even a wholly unsuspected brain tumor may be disclosed.

A further reason for the more frequent use of air encephalography is its therapeutic effect, particularly in relation to posttraumatic headache. While this aspect is not pertinent to the subject under discussion, nevertheless I cannot refrain from mentioning it, since it might be the one and perhaps only, means of dealing effectually with a compensation case. In my experience, oxygen encephalography has played a real part in curing posttraumatic discomfort, especially headache.

One other special examination, that is, lumbar puncture, should be noted. In my experience, its value is remote in the type of case under discussion, but it might give such information as a high total protein, or in occasional instances, might yield a positive Wassermann reaction. Certainly if the latter were true, one would want to make repeated examinations and, in addition, a careful search for other evidences of syphilis.

3. *Criteria as to longevity.*—In gauging actual life expectancy after a head injury, any possibilities which would lead later to an intracranial septic condition are of extreme importance. A persistent, chronic otitis media following a fracture involv-

ing the middle fossa is perhaps the most common such condition. This might eventuate in meningitis or a brain abscess, and in patients with suspicious symptoms, one should look for signs of the latter, such as aphasia, homonymous hemianopsia, central facial weakness, nystagmus and ataxia. In war injuries, with intracranial retained foreign bodies, a brain abscess may develop many months or even years after the injury. Another but rare condition which if untreated almost inevitably leads to subsequent meningitis, is a persistent cerebrospinal rhinorrhea or an intracranial aerocele from injuries involving particularly the ethmoid sinuses. These conditions will always be extremely serious, but the use of sulfonamides may do much to lessen fatalities.

A second condition of importance in judging life expectancy is chronic subdural hematoma. As a rule, within a few weeks after a head injury, these extensive encapsulated clots give some evidence of their presence; however, in other instances, several months may elapse before symptoms appear. Often the injury may have been thought originally to be trivial. The commonest symptoms are unforeseen mental changes of varying character and headaches which may become extreme in intensity, but almost any combination of neurologic signs may be present.

Another occasional complication of cranial trauma is damage to an intracranial blood vessel, the wall of which may become weakened and result in subsequent rupture or possibly a true post-traumatic aneurysm. If such a condition is suspected, diagnosis may require an arteriogram. This is accomplished by making roentgenologic examinations of the skull while injecting thorotrast into the common carotid artery.

Convulsions, which are one of the recognized sequelae of craniocerebral injuries, may play an important role in life expectancy. No conclusive figures can be given regarding the incidence of convulsions, since there is so much that is conflicting in published statistics. The percentages vary from about 5 per cent in a large series of gunshot injuries with or

without penetration of the dura (Turner, 1923, quoted by Grant) (5) to over 30 per cent in a smaller but similar and carefully studied series recently reported by Ascroft (6). The latter included purely scalp wounds, as well as those of a more serious variety, and it seems astounding that 24 per cent of the patients having only scalp wounds developed fits. Ascroft showed particularly the importance of sepsis in head wounds as related to the subsequent development of convulsions.

In Stewart's series of cases (also quoted by Grant), which was likewise studied with regard to post-traumatic epilepsy, the incidence was 8 per cent for all kinds of skull injury. Still another much quoted series is that of Wagstaffe (7), giving an incidence of 18.7 per cent for wounds with dural penetration and 1.6 per cent for those without.

From the purely medical aspect, it is sometimes exceedingly difficult to decide whether the convulsions were actually caused by the head injury, or whether the injury was merely a contributory factor in a person whose family history gave evidence of epileptic tendencies. This is particularly difficult in the case of young children. However, from the insurance standpoint, I should suppose that it would make little difference whether the head injury was the principal or the contributory factor. The liability is undoubtedly there, unless it could be shown that fits had preceded the head injury, or perhaps that the hereditary taint was so strong as to make convulsions almost certain. The latter would be exceedingly hard to prove.

The danger to life in an individual suffering from convulsions must be obvious. Any fall during a seizure may result in serious cranial or intracranial damage. Death might occur if the patient had a convulsion while driving a car or when near some form of machinery, when in swimming over his depth, or when on a ladder or scaffold. Rarely the patient may die as the result of the convulsion itself, possibly from a cerebral hemorrhage or respiratory failure.

Finally, the patient's emotional state and background with the resultant upset due to head trauma, must be taken into consideration on the basis of possible suicide. I am not able to evaluate this factor personally, but merely mention it as one of the contributing factors.

4. *Criteria as to disability.*—In this category one must consider not only such conditions as described under criteria for longevity, but also a wide variety of other factors.

The severity of the injury and its residual evidences should be determined as accurately as possible, together with other information to which reference has been made under historical data, including the occupation of the patient or what he was fitted for by training or by mental and physical capacity. A rough estimate as to the expected length of disability may be made on the basis of the length of posttraumatic amnesia. This, along with many other aspects of diagnosis and prognosis in head injuries, has been the subject of intensive study by a group of British neurologists and neurosurgeons, notably Denny-Brown and Ritchie Russell (8) and Lewis (9), in addition to Cairns, Symonds and Jefferson, whose work has been mentioned. Many of these authors stress particularly in regard to prognosis, the length of time which has elapsed between a head injury and consultation with a neurologist, believing that the longer this interval the more firmly has the patient's fixation on his discomforts become.

The advantages of an early neurologic or neurosurgical consultation, together with early and continued proper treatments for patients who have suffered a head injury, can hardly be overestimated. It is along this line in relation to war time casualties and in civilian injuries thereafter that you, as medical directors of insurance companies, may play a most important role in the months and years to come. Such early and intelligent treatment would be of the utmost advantage to your clients, in addition to your own financial saving.

Again, in any consideration as to prognosis, the type of disability from which the patient suffers is paramount. Is it

purely a matter of headache, dizziness, nervousness and other similar symptoms of the so-called posttraumatic state, or are there objective evidences of cranial nerve palsies, hemiparesis, aphasia, convulsions or possibly visual changes? Among the former, Symonds stresses the frequency of olfactory impairment which is often overlooked. Other fairly common cranial nerve injuries are those to the sixth, third and seventh nerves. At least six months to a year should be allowed for possible spontaneous recovery in these cases.

In evaluating any or all of these factors, electro-encephalographic studies made as soon as practicable after the injury and repeated at intervals of two to four weeks are becoming increasingly important. The immediate record may present definite abnormalities, but as the patient's condition improves, the record becomes more and more normal. The actual details of the readings are not pertinent to the subject under discussion, but they must, of course, be interpreted by one who is thoroughly familiar with this branch of neurologic diagnosis.

Before passing final judgment as to the degree of disability from any head injury, it may be necessary, or at least wise, to give the patient a period of trial in order to see how he reacts to intensive therapy and an increasingly active regimen. This should include physical therapy, intellectual and recreational therapy and competitive pursuits in order to determine his cooperation. Rarely, during this period or at some earlier time, operative measures may have to be instituted. I refer particularly to air encephalograms, occasional trephine openings if a chronic subdural hematoma is suspected, or still more rarely, a decompression.

In addition to the examinations mentioned, one must bear in mind that in some cases consultation with a trained psychologist is of value in forming a correct opinion. Such a report should not be a simple statement of the patient's intelligence quotient, but a considered estimate of his character in all of its various aspects. This report should be given its due weight along with the reports of other consultants such as

ophthalmologists, otologists, or any others with training in specialized fields.

Finally, after all the available data are collected and after all methods directed toward the permanent recovery of the patient have been given a fair trial, judgment as to the residual disability and its compensation should be made by some physician with wide experience in matters of this kind. Preferably, such a person would be a neurologist who had had ample training not only in his own specialty, but in psychiatry and in neurosurgery. Fortunately, for the purposes of decisions to be made in the future, individuals of this type are being trained in ever increasing numbers, thanks to the evolution, — or I might say revolution through which neurology as a whole has been passing during the last decade.

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PRESIDENT WENSTRAND—Dr. Peter G. Denker, Assistant Medical Director of the Equitable Life Assurance Society, will continue the discussion. Dr. Denker!

DR. DENKER—I feel it is almost presumptuous on my part, with an authority of Dr. Horrax's standing having addressed us here, and given us such a good, comprehensive picture of the problem, to try to say anything at all in amplification. My only reason for injecting a few comments is that possibly I can present some of the problems from a slightly different aspect, as they are presented to us in the field of selection and in disability evaluation.

One of the most important things on which we lay emphasis in trying to evaluate the severity of head injuries is, as Dr. Horrax mentioned, the degree of unconsciousness that the patient sustained at the time of injury. It is extremely difficult for us, especially in cases for rather small amounts of insurance that have been examined by general practitioners rather than by neurologists or neuro-surgeons, to obtain all the data we would like to have, and that we really should have, in a more intelligent evaluation of the case, so that the duration of unconsciousness which the patient sustained with his head injury, is a good rough criterion.

We have subdivided the cases of head injury into, briefly, three categories in attempting to underwrite these cases. In the first group, the mildest group, we have limited the unconsciousness to a period of a few minutes, up to fifteen minutes. It is a purely arbitrary selection, but we feel that in those cases, the brain injury, which is the more important thing, rather than the injury to the skull vault itself, was probably minimal. So that, for six months, we do not consider these cases for insurance.

We feel six months is a fair period of time in which to see whether any complications may evolve—possibly a subdural hematoma, or epileptic attacks due to some sort of scarring in the brain, etc. If, after that period of time, however, they pass a normal neurological examination, we will usually accept them at standard rates. We will, however, decline them for disability or double accident features until a little longer

period of time has elapsed. After the second year, we are likely to be more lenient and even grant them the features.

In the second group of cases, are those who have been unconscious for a few hours. Again, we arbitrarily draw a line there, as a convenient guide, at six hours. If they have been unconscious from fifteen minutes to six hours, we put them into our Class B. We are a little more drastic in our treatment in these cases, and require a little longer period of time before we insure them. We usually wait at least a year. For the second and third years, we give them mild substandard ratings, and after that, if they still persist in good health, show nothing on neurological examination, and we cannot find anything from the inspection data (to which I will come in a moment), we issue a standard policy.

The last group, the more serious injuries, falls into two categories. Either they have been unconscious for a longer period of time than six hours, making us feel that the brain damage was more severe, or they may have had depressed fractures of the skull, infections of the skull with underlying scarring, or have been operated upon for a subdural hematoma, where the risk of future scarring is greater. That latter group we treat most severely, and decline them for at least a year. In the second year, we give them more highly substandard ratings than the uncomplicated cases, unconscious for a few hours. Until the end of the fifth year, we still issue substandard ratings at diminishing rates on these cases. If after five years, they have gone along well, show nothing on neurological examination (we are more likely to ask for x-rays of the skull or other data in this sort of case), we will issue a standard policy.

The remarks I have just made hold true for people who show no sequellae of head injury on examination. Once in a while, we have an applicant who has been operated upon and who may show a residual hemiparesis. If that has gone along for a period of years, has not progressed, and he has not had any epileptic seizures, or anything of importance

on neurological examination, we are likely to issue a substandard policy on the basis of the amount of deformity that has persisted.

We do not feel that a hemiparesis of that sort is analogous to a hemiparesis that may be the result of vascular disease, brain tumor or various other intracranial conditions.

I would like to stress one or two other points which I think are of practical importance, as the problem is presented to us in the underwriting sphere. Dr. Horrax mentioned the importance of the psychiatric evaluation of these organic head injury cases. We feel that is extremely important. It makes a good deal of difference as to *who* received the injury, and whether we are getting a truthful picture of the case. As so often happens, we do not get the whole story, so that we have become accustomed to rely, to a great extent, on the inspection reports in evaluating this problem in addition to our medical data.

Especially in the vague realm of personality changes do we find these reports helpful. Often, an examiner will go over the applicant neurologically, find reflexes normal, nothing wrong with his pupils or fundi or sensation, and give him a clean bill of health medically, and then we find on the inspection report that, since this man sustained the head injury, there has been a marked change in his personality. This not only holds true in evaluating the degree of disability, but also in estimation of his insurability.

Such a personality defect may be the only residuum of a head injury, so that the people who have been in contact with him for many years, who know him well, who have been working with him in his place of business or are associated with him around his residence, are likely to give us information in that sphere which is quite important in the evaluation of the problem.

We do not resort to air studies in our selection, but when air studies have been done, and ventricular deformities are

found, we have to assess them in relation to the severity of the impairment. If they are severe, we do not make any insurance offer.

The electroencephalogram is a more recent addition. We don't know much about it. It is just beginning to be worked out and its scientific evaluation, as Dr. Horrax brought out, is not yet complete. We feel that this will be an important addition in the disability evaluation of a problem.

One of the very important problems in disability evaluation is the question of the organic versus the functional post-concussion symptoms. In most of those cases, on a routine neurological examination, very little is discovered. Still, we know from experience, from encephalographic studies in these cases, from the electroencephalogram recordings, that many of these cases that we have been too glibly labeling as traumatic neurosis have underlying cerebral pathology.

If we feel convinced, with our present means of evaluating this problem, that there is no organic damage underneath, or, at least, if all the data that we have do not indicate that there is, I believe it is very important in these disability cases to try to settle such a case early. A post-traumatic neurosis which, unfortunately, is called post-traumatic, though often the trauma was merely the factor, is just like any other neurosis in relation to disability income. The more income they have, the more we pay them monthly, so to speak, as a price for their being sick, the longer the neurosis will last. That is probably very trite and you have heard that comment made time and time again, but it is especially important in this group of head injuries. So that, if all the data on hand shows nothing organic, I always recommend to the disability department that we try to settle that case, preferably by means of a lump sum settlement and as early as possible.

Lastly, I would like to ask Dr. Horrax if he could possibly give us here a little help, on one point. I heard Dr. Leary at the Academy of Medicine the other night talk about some-

thing new that Dr. Penfield was using in these head injury cases, where he had to resort to operative procedure. He said he had some sort of membrane made from the amnion, which they inserted. It looked like a little piece of cellophane. They inserted that underneath the wound of the skull, overlying the dura, the idea being to prevent adhesions.

Naturally, if a thing like that does help in preventing cortical scars, it would be of great help to us in future prevention of epilepsy. I was wondering if Dr. Horrax had had any experience in that field. I know it is something new.

I also would like to know whether by any chance he has had any success with a new drug, prostigmine, in the relief of of some of these post-concussion syndromes. Dr. Malone, in a recent article in the Journal of the American Medical Association, described a group of cases in which that drug was remarkably effective in the relief of the two outstanding symptoms in these cases—headaches and dizziness. We have had very little experience with it so far, and I would be greatly indebted to Dr. Horrax to learn more about it.

PRESIDENT WENSTRAND—If Dr. Horrax would be so kind as to answer other questions, perhaps there are some from the members. Is there anyone who has any questions to ask? If not, Dr. Horrax, will you answer Dr. Denker and close the discussion?

DR. HORRAX—I may say I am very much indebted to Dr. Denker for this very valuable amplification, discussion and addition to what I had to say, in which, of course, I am thoroughly in agreement.

As to the amniotic membrane, we have used that to some extent. I have no specific data as to whether it actually will prevent convulsions, but I can tell you this, I think, safely because I have had a talk with Penfield about it, that he was very enthusiastic about this amniotic membrane at first, but since his early experiences, I believe he has found that just about as many adhesions form with amniotic membrane as

with other kinds of tissue, and it is possible that Dr. Leary may have meant that he was working with some other type of tissue at the present time, which I think he is. But as far as I know, the amniotic membrane eventually causes just as many adhesions as other things, like cellophane or other tissues, the smooth tissues that one may use in the brain.

We have tried various things like that — gutta percha, for example — but they all allow adhesions to form.

So far as prostigmine is concerned, I have not had any personal experience with that. The post-traumatic headaches that we see at the clinic are given such medical treatment as they may need by our medical department at first, and if they don't cure them, they turn them over to us to see what we can do about them surgically. We may have, of course, cases which are nonsurgical which we treat by other means first, too, but I personally have not had any experience with prostigmine.

I feel that this, like other things, is most enthusiastically done by those who have the earliest and first experience with it, and I think it certainly deserves a trial but I have had no personal experience with it.

PRESIDENT WENSTRAND—Dr. Frank Norman Wilson needs no introduction to this Association for we all remember him and his clear and instructive address on Electrocardiography in 1937. I present to you again Dr. Wilson, Professor of Medicine at the University of Michigan Medical School, who will tell us about "The Precordial Electrocardiogram" and other things new in this branch of cardiology.

THE PRECORDIAL ELECTROCARDIOGRAM*

By Frank N. Wilson, M. D.

in collaboration with

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Introduction. Considerable progress has been made in the standardization of precordial leads, but there is still a great diversity of opinion as to how many leads of this kind should be taken, as to what precordial points should be explored, and as to the proper location for the remote or reference electrode. The multiplicity of leads still in use and still being recommended is the source of much confusion and continues to retard progress in the setting up of adequate normal standards and satisfactory diagnostic criteria applicable to the interpretation of the precordial electrocardiogram. It is not, therefore, surprising that there has been a persistent demand that a single standard method for taking precordial leads be established. Unfortunately, it is not easy to meet this demand for there is no generally accepted method for determining which leads are likely to prove most and which least useful, and there are very great differences of opinion as to whether the decisions required should be made on empirical grounds alone, or if not as to what general principles should be taken into consideration.

It seems to me that it would be less difficult to solve this problem if there were a more widespread and clearer understanding of the nature of precordial leads, the relations between them and leads of other kinds, the considerations which

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led to their use, the advantages which have made them popular, and the purposes which they may be expected to serve. I propose, therefore, to discuss the precordial and other special leads which have been used with much satisfaction in my own laboratory. I want to emphasize the principles upon which they are based and to call attention to the kind of information that may be obtained by their use in cases of bundle branch block, ventricular hypertrophy, and myocardial infarction. It is in these conditions particularly that the standard limb leads have been found inadequate.

It is true that much of our present knowledge of the human electrocardiogram has been gained by the correlation of electrocardiographic data and clinical or post-mortem observations. Nevertheless, electrocardiography can hardly be considered a purely empirical science. Its physical and physiological foundations rest upon a large body of critical experimentation dictated by well-constructed hypotheses. These foundations were laid and the more fundamental principles upon which the interpretation of electrocardiographic records is based were established before the string galvanometer was introduced into clinical medicine. The more important advances made since that time have come about in the same way as those which preceded them. Few major electrocardiographic problems have been solved by clinical observations alone. For this there are excellent reasons. Our basic knowledge of action currents was acquired by means of direct leads from the tissues under investigation. Unfortunately, is was not possible to study the action currents of the human heart by this method and indirect leads were necessarily employed for this purpose. The standard limb leads now in universal use have become indispensable and they are entirely adequate for most of the purposes which clinical electrocardiograms can be expected to serve. They are not, however, capable of furnishing detailed information of the sort obtained when the surface of the exposed heart is explored by means of direct leads. Experi-

ments on animals in which such leads have been employed have consequently continued to play a very important role in the solution of our most difficult electrocardiographic problems. We have not, however, been able hitherto to use all the knowledge acquired in this way to the best possible advantage, because the difference in character between the limb leads used in man and the direct leads used in animal experiments has made it difficult to correlate our clinical and experimental observations. Precordial leads of the type now widely used are much more closely related to direct leads than to the standard limb leads. It is precisely for this reason that they are of such great value. They not only detect lesions which are beyond the range of the standard limb leads, but furnish data that can be directly compared with the data obtained by the use of epicardial leads in animals.

Unipolar direct leads. For the study of cardiac action currents not all kinds of direct leads are equally satisfactory. In their extensive and detailed study of the course of the excitatory process over the ventricular muscle Lewis and Rothschild (1) found that it was difficult or impossible to interpret the records obtained when both electrodes were in contact with the ventricular surface. When leads of this kind were used the cardiac impulse spreading through the ventricular wall from within outward often reached both electrodes at approximately the same time. The deflections recorded under these circumstances were of complicated and variable form and gave no reliable clue to the time of arrival of the impulse beneath either electrode. When, on the other hand, a small electrode in contact with the epicardium was paired with an electrode on the chest wall the *QRS* complex almost invariably displayed a large and very abrupt deflection which was easily recognized, and was found to coincide with the activation of the muscle in contact with the exploring electrode. The name "intrinsic deflection" was therefore assigned to it. The other deflections of the *QRS* group, which were attributed to the excitation of muscle more distant from

this electrode were referred to as "extrinsic deflections", but their origin was not studied in detail.

Because of the character of the laws which govern the distribution of electric currents in three-dimensional conductors such as the body (2) the magnitude of the potential variations produced by the heartbeat at any point outside the heart itself rapidly diminishes as the distance from the epicardial surface increases. In the course of a cardiac cycle the variations in potential at a point on the ventricular surface are in the neighborhood of thirty times as large as those displayed by the arms and legs. The deflections of leads from the ventricular surface to some distant part of the body, such as those employed by Lewis and Rothschild, represent the potential variations of the electrode in contact with the heart; the potential variations of the distant electrode are negligible by comparison. It is convenient to use the term "unipolar" to designate leads of this kind in which one electrode is almost completely indifferent, and the term "bipolar" to designate leads like the standard limb leads in which the two electrodes are approximately equidistant from the heart and may, in general, be expected to undergo potential variations of about the same magnitude.

The electrocardiograms reproduced in Fig. 1 were obtained by means of unipolar direct leads from the exposed heart of a dog. Six points on the anterior surface of the ventricle, easy to identify later by their relations to the vessels or other surface markings, were selected and from these control curves were taken with a small exploring electrode. Points *7*, *6* and *8* were on the right ventricle; the first was on the thickest part of the wall near the atrioventricular groove and the other two were in the central region close to the trabeculated area which overlies the attachment of the anterior papillary muscle. Points *3*, *1* and *2* were on the left ventricle; the first was very close to the junction of the free wall and septum, the second near the left margin of the heart and the third very near the apex. After the control records had

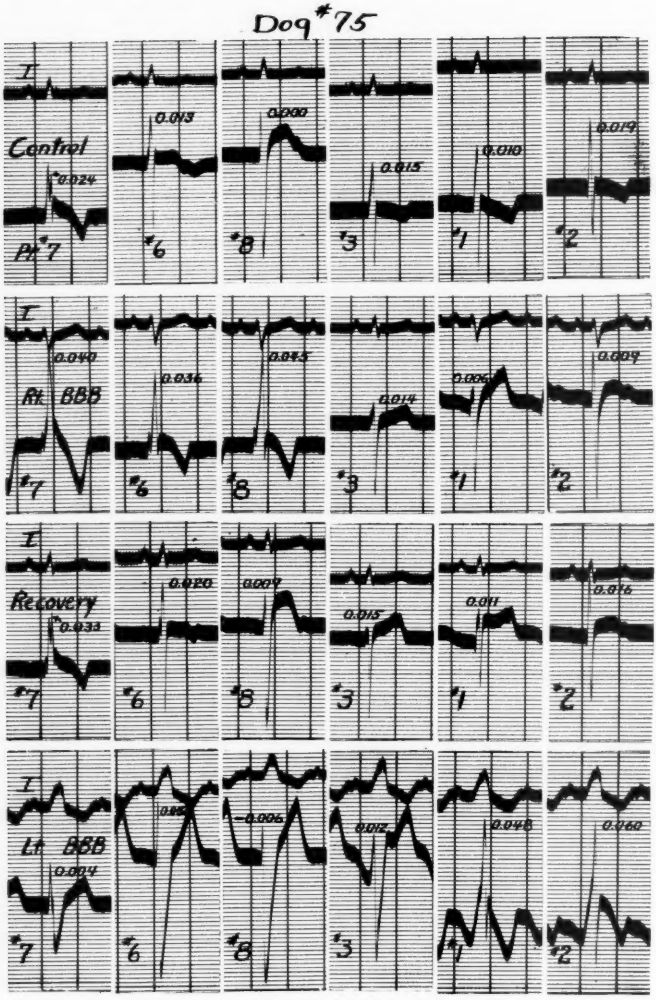


Fig. 1

Unipolar direct leads from six points on the exposed ventricular surface of a dog's heart.

been taken temporary right bundle branch block was produced by pressing upon the right side of the septum above the anterior papillary muscle with the back of a small knife thrust through the wall of the conus arteriosus just below the pulmonary valve. A second set of curves was taken immediately after the knife was withdrawn, and a third set when the right bundle branch had recovered almost completely. Left branch block was then produced by making a cut on the septum just beneath the aortic valve, and a fourth set of records was made. Standard lead I was taken simultaneously with each direct lead. The intrinsic deflection is easily identified in all of the curves except the first and third from point *7*; it is the sudden downward movement which begins with the peak of *R*. The beginning of this deflection, and therefore the peak of *R*, marks the time of activation of the subepicardial muscle in contact with the exploring electrode. Whether the activation of this muscle and the beginning of the intrinsic deflection coincide exactly or only approximately need not concern us here. It will be noted that in the leads from the right ventricle the peak of *R* fell very late in the *QRS* interval when right branch block was present and at the very beginning of this interval when left branch block was present. In the leads from points *1* and *2* on the left ventricle these relations are reversed; the peak of *R* fell late in the *QRS* interval when left branch block was present and early when right branch block was present. No curve was obtained from point *3* while right branch block was present for the form of the ventricular deflections of lead I shows that when the second tracing was taken from this point the right bundle branch had already recovered its conductivity. In the lead from point *3* taken after left branch block was produced the peak of *R* was not later than before. This point was very close to the septum and in our experience the intrinsic deflection in leads from this region has seldom been much delayed either by left or by right branch block.

When the cardiac mechanism is normal the intrinsic deflection is always earliest in leads from the trabeculated

region, as in the present instance (point *8*). It is only a very little later in leads from the central region but considerably later in leads from points near the atrioventricular groove (point *7*) and from points on the conus arteriosus. The earliest intrinsic deflections found on the left ventricle usually occur in leads from the apex (point *2*); the latest in leads from the lateral part of the free wall toward the base. This region was not studied in the experiment under consideration. The intrinsic deflection is normally considerably later over the surface of the left ventricle as a whole than over the surface of the right. This difference is attributable to the greater thickness of the left ventricular wall, for in general the latest intrinsic deflections are found where the muscle is thickest.

Origin of the QRS deflections. The origin of the more conspicuous and more constant components of the various kinds of ventricular complexes seen in unipolar direct leads is clearly understood. It must be emphasized that although the excitation of the muscle in contact with the exploring electrode produces a much larger and much more sudden fluctuation in the potential of this electrode than the excitation of any equal mass of muscle at a greater distance from it, every unit of ventricular muscle, without exception, produces action currents which contribute to the form of these complexes. It is obvious that those components of the *QRS* complex which precede the intrinsic deflection must be attributed to the excitation of muscle which passed into the active state ahead of the subepicardial muscle beneath the exploring electrode, and that those components which follow the intrinsic deflection must be ascribed to the excitation of muscle that was still in the resting state when the muscle under this contact became active. The peak of *R*, therefore, separates the *QRS* components attributable to muscle activated before from the *QRS* components attributable to muscle activated after the cardiac impulse reached the muscle in contact with the exploring electrode.

The resting cardiac muscle fiber is surrounded by a "mem-

brane" which has a very low electrical conductivity. Across this membrane there is an electromotive force which makes its inner surface strongly negative in comparison with its outer surface. The passage of the excitatory process along the fiber is associated with a sudden and very great increase in the conductivity of the resting membrane and a sudden decrease in the electromotive force (Cole and Curtis 3); in other words with the sudden development of a short circuit between the inside and the outside of the fiber. Current flows out of the resting part of the fiber adjacent to the short circuit and re-enters the fiber through the short-circuited region. The effect in the medium outside the fiber is the same as if the cardiac impulse were immediately preceded by the positive pole and accompanied by the negative pole of a battery. In other words, we may say that across the boundary between active and resting muscle there is an electromotive force which makes the potential of points toward which the excitation wave is advancing positive and the potential of points which lie behind it negative.

The cardiac impulse spreads through the free walls of the ventricles from within outwards and normally enters the septum from both sides simultaneously. Throughout its course it is traveling away from the ventricular cavities, and these are continuously on the negative side of all the boundaries between active and resting muscle. Consequently the potential of both cavities is negative throughout the *QRS* interval (4) except in those instances in which one side of the septum becomes active before the other, in which case the ventricular cavity on the side activated last almost always displays initial positivity.

Electromotive forces distant from two neighboring points have approximately the same effect upon the potential of both. It is, then, clear that no cardiac electromotive force except that generated by the muscle between them is capable of producing a large difference of potential between two points which are separated solely by the thickness of the ven-

tricular wall. If the potential of the adjacent part of the ventricular cavity is negative the potential of an epicardial electrode can be positive only while the excitation wave is passing through the part of the ventricular wall upon which it rests. In taking unipolar leads the galvanometer connections are so made that positivity of the exploring electrode produces an upward deflection in the completed record. The *R* deflection in unipolar leads from the ventricular epicardium of the normal heart must, therefore, represent the electrical forces produced by excitation of the muscle between the exploring electrode and the ventricular cavity. The total voltage of these forces is, of course, not measured by the height of the *R* deflection, but by the difference in potential between the endocardial and the epicardial surfaces of that part of the ventricular wall in contact with the exploring electrode. In bundle branch block most, if not all, of the septal muscle is activated from the contralateral side, and the potential of the homolateral cavity is positive until the cardiac impulse has passed through the septum. Under these circumstances the *R* deflection in leads from the outer surface of the delayed ventricle ordinarily displays two peaks. The part of this deflection which precedes the division is due to activation of the septum and represents positivity of the ventricular cavity which is transmitted through the inactive ventricular wall; the part which follows the division is due to activation of the ventricular wall adjacent to the electrode. When this second peak of *R* is written the ventricular cavity is negative.

When the excitatory process reaches the muscle in contact with an epicardial electrode the boundary between resting and active muscle disappears from that part of the ventricular wall upon which the electrode rests. The potential of the electrode then becomes the potential of the adjacent part of the ventricular cavity. The sudden drop in the potential of the electrode when the electromotive force across the wall beneath it disappears is responsible for the intrinsic deflection. The total length of this deflection is an approx-

imate measure of the voltage across the ventricular wall when the cardiac impulse arrived at the epicardial surface.

If the excitatory process is still spreading through some part of the ventricular wall when the intrinsic deflection occurs, the negativity of the ventricular cavity outlasts this deflection and an *S* wave is inscribed. If, on the other hand, the muscle in contact with the exploring electrode is the last ventricular muscle to pass into the active state the ventricular cavity is at zero potential when the intrinsic deflection is complete and there can be no *S* deflection.

If the subendocardial muscle of some part of the ventricular wall passes into the active state earlier than the subendocardial muscle which lies between the exploring electrode and the ventricular cavity the initial negativity of the cavity is transmitted to the electrode and a *Q* deflection occurs. If the cavity is initially positive because of circumstances that I have already mentioned there can be no *Q*. Since late activation of the subendocardial muscle is likely to be associated with late activation of the subepicardial muscle of the same region *Q* deflections are most common in those leads in which the intrinsic deflection occurs late in the *QRS* interval, and in which, therefore, *R* is tall and *S* is small or absent.

In summarizing my remarks on the interpretation of the *QRS* deflections of unipolar direct leads from the ventricular surface I may point out that the character of these deflections depends mainly upon the time at which the muscle in contact with the exploring electrode becomes active in relation to the beginning and to the end of the *QRS* interval. If this muscle becomes active very early in this interval the *R* deflection is narrow and usually small and the *S* wave broad and usually deep. If it is activated very late in this interval *R* is tall and broad, *S* small and narrow and a *Q* wave often present. If it is activated at the very end of the *QRS* interval *S* is absent, but there may be a conspicuous *Q*. As the time of activation of the muscle beneath the exploring electrode approaches the beginning of the *QRS* interval *R*

becomes smaller, *S* larger and *Q* less frequent. As it approaches the end of the *QRS* interval *R* becomes larger, *S* smaller and *Q* more frequent. In this discussion I have, of course, not considered many of the perplexing details of the *QRS* complex which may be encountered in unipolar direct leads as well as in leads of other kinds. Some of these cannot be interpreted satisfactorily until more data are available than at present.

Unipolar precordial leads. The potential of every extracardiac point inside the body is determined by the potential over the heart's outer surface. The potential variations of every element of the epicardial surface contribute in some measure to the potential variations of an electrode placed upon the precordium. But the magnitude of the contribution made by the potential variations of any given surface element is large if its distance from the electrode is small and vice versa; it varies, in fact, roughly as the inverse cube of this distance. For this reason the potential variations of a precordial electrode are determined to a very large extent by the potential variations of the elements of ventricular surface nearest it. If all or the great majority of these elements simultaneously display potential variations of the same sort the potential variations of the electrode are of the same character though very much smaller. If, on the other hand, the various elements that are more or less equidistant from the electrode display potential variations of many different kinds the potential variations of the electrode represent a mixture which may not resemble any of its components very closely.

Since the conditions last mentioned rarely exist it is almost always possible by moving an electrode step by step across the precordium to determine the nature of the potential variations of the various parts of the anterior ventricular surface, provided that the potential variations of the exploring electrode are accurately recorded. In 1929 we began to use multiple precordial leads of this kind to ascertain which of the two ventricles first became active in the classical varieties of bundle branch block. In our first experiments the pre-

cordial electrode was paired with an electrode on the left leg. The data obtained in this way were not entirely conclusive because the components of the *QRS* complex due to potential variations of the precordial electrode could not be distinguished with certainty from those due to potential variations of the leg electrode.

In man, the potential variations of the precordium are ordinarily three to five times as large as those of the extremities or of the back. When a precordial electrode is paired with an extremity or dorsal electrode the general outline of the ventricular complex is, therefore, determined to a very large extent by the potential variations of the former, and is much the same regardless of whether the latter is placed upon the right arm, the left arm, the left leg, or the left infrascapular region. But in leads of this kind the potential variations of the reference electrode do exert a very considerable influence upon the details of the form of the ventricular complex, and make it impossible to utilize the simple principles applicable to the interpretation of the deflections of *unipolar direct leads* in the analysis of the records obtained.

Many seem to believe that precordial leads are merely additional leads comparable in all respects to the standard limb leads. The practice of taking a single precordial lead and calling it lead *IV*, a practice which we have never followed, is no doubt partly responsible for this entirely unjustifiable attitude. There is no limit to the number of pairs of points on the trunk which might have served as locations for the electrodes if the "cut and try" method had been used to find the additional leads most likely to prove helpful in those situations in which limb leads had been found inadequate. Precordial leads of the kind under consideration were selected for this purpose because observations dictated by theoretical considerations showed that they are in reality semidirect leads from the anterior ventricular surface, capable within certain limits of serving the same purposes as direct leads

from the ventral wall of the exposed heart. They cannot serve these purposes, however, unless the potential variations of the reference electrode are made negligibly small in comparison with those of the exploring electrode. The extent to which the character of the potential variations of the precordial electrode is obscured by the potential variations of the remote electrode determines the degree to which the advantages gained by placing one electrode upon the precordium have been surrendered.

Considerations of this kind led us to devise an artificial ground which reduces the potential variations of the remote electrode to a minimum and makes it possible to obtain essentially unipolar leads from the precordium, from the esophagus, from the extremities, and from other parts of the body. This artificial ground consists of a central terminal which is connected through three equal resistances of 5000 ohms each to electrodes on the right arm, left arm, and left leg (5). The effect of this device is to balance out those electric forces responsible for differences in potential between these extremities and consequently for the deflections of the standard limb leads. It is easily demonstrated that at every instant the potential of the central terminal must be equal to the algebraic mean of the potentials of the three extremity electrodes. On the basis of the assumptions upon which Einthoven's equilateral triangle is founded it has been shown that those components of the heart's resultant electromotive force which are parallel to the plane defined by this triangle have no effect whatever upon the potential of the central terminal. The effects of those components which are perpendicular to this plane cannot be accurately predicted on theoretical grounds. The experiments of Eckey and Fröhlich (6), Burger (7), Burger and Wuhrmann (8), and of others (9) have, however, demonstrated that the largest potential variations of the central terminal do not ordinarily exceed 0.3 millivolt. The potential of this terminal is, therefore, so nearly constant that for practical purposes we may consider it zero throughout the cardiac cycle. We shall

then consider all leads unipolar in which an exploring electrode is paired with an artificial ground of this type.

When we began to use precordial leads in our studies of human and experimental bundle branch block it soon became evident that the most satisfactory way to obtain semidirect leads from both ventricles was to move the exploring electrode across the precordium along a line roughly perpendicular to the anterior interventricular sulcus and extending from a point at least as far to the right as the right margin of the sternum to a point well beyond the left border of the heart. We have changed our original method of taking precordial leads only as regards the number and the locations of the points explored, which have undergone minor modifications from time to time. We now take unipolar leads from each of the six points specified by the Committee of the American Heart Association for the Standardization of Precordial Leads (10). In accordance with the recommendations of this committee these leads are designated V_1, V_2, V_3, V_4, V_5 and V_6. The first of these six points is at the right margin of the sternum in the fourth interspace and the second on the left sternal margin at the same level. The remaining points lie on a broken line extending from the second point to the apex beat and thence around the left side of the chest at the apical level. When the apex beat cannot be located this line is drawn from the second point to the intersection of the left midclavicular line and the fifth interspace and is continued around the chest at the level of this intersection. The third point is midway between the second and the left midclavicular line, the fourth in the midclavicular line, the fifth in the anterior axillary line, and the sixth in the midaxillary line. In addition to these six standard leads we usually take a unipolar lead from the tip of the ensiform cartilage (lead V_E) and often take unipolar leads from the posterior axillary line (lead V_7), from the left infrascapular region (V_B), and from other points.

In the precordial electrocardiograms of normal subjects the ventricular complexes of the leads from the right side of the

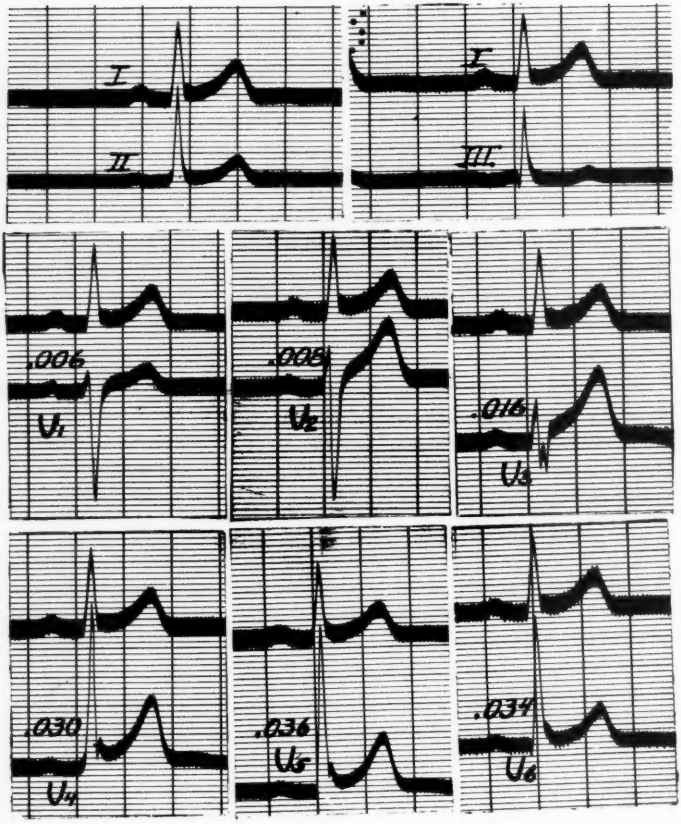


Fig. 2

The precordial electrocardiogram of a normal subject. The decimals marked on each record give the time of the intrinsic deflection with reference to the beginning of the *QRS* interval in lead *I*, which was taken simultaneously with each precordial lead. (Reproduced from an article by Wilson in Stroud's "Diagnosis and Treatment of Cardiovascular Disease", by permission of the F. H. Davis Company, Philadelphia).

precordium are strikingly similar to those obtained by placing the exploring electrode directly upon the thinner parts of the anterior wall of the dog's right ventricle (Fig. 2). In these complexes the *R* deflection is small and narrow and *S* is deep and relatively broad. The peak of *R* occurs early in the *QRS* interval and is followed by a large, abrupt downward movement which obviously corresponds to the intrinsic deflection of direct leads. The ventricular complexes of the leads from the left side of the precordium are similar to those seen in direct leads from the thicker parts of the anterior and left lateral walls of the dog's left ventricle. The *R* component of these complexes is tall and relatively wide and it is often preceded by a *Q* and followed by an *S* deflection. The peak of *R* which marks the onset of the intrinsic downstroke is on the average about *0.02* second later in leads from the left side of the precordium than in leads from the right side (11). In precordial leads this downstroke is not nearly so sudden as in direct leads and is, therefore, somewhat more difficult to identify with certainty when the contour of the ventricular complex is complicated or unfamiliar.

Between those parts of the precordium which yield small *R* and deep *S* waves and those which yield *QRS* complexes of the opposite kind there is a zone which yields complexes that are intermediate in form or represent a combination of the deflections obtained at points farther to the right and those obtained at points farther to the left. This zone varies from subject to subject both in location and in width, and is probably not absolutely constant in either particular in one and the same subject. The ventricular complexes of leads from this transitional zone may display notching and other peculiarities of form. They represent a mixture to which the potential variations of those parts of the right ventricle and the potential variations of those parts of the left ventricle which are least distant from the exploring electrode contribute in approximately equal measure.

The minimal, maximal, and mean voltages of *Q*, *R*, *S* and *T* in precordial leads V_1, V_2, V_3, V_4, V_5 and V_E and in unipolar

TABLE I

THE SIZE OF THE VENTRICULAR DEFLECTIONS IN THE STANDARD AND SPECIAL LEADS (MEASUREMENTS GIVEN IN TENTHS OF A MILLIVOLT)

	Q				R				S				T				RS			
LEAD	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
I	0	1.5	0.33	0.45	1.5	19.4	6.81	3.27	0	5.0	1.67	1.35	1.0	5.5	2.21	1.05	3.0	20.6	8.63	3.20
II	0	2.0	0.43	0.61	4.0	22.0	11.99	4.39	0	8.0	1.53	1.92	1.0	6.0	2.97	1.10	8.0	23.0	13.76	3.72
III	0	2.0	0.54	0.60	1.2	18.0	8.50	4.33	0	13.0	1.27	2.40	0	3.0	1.49	0.75	3.2	18.0	10.12	4.10
V_R	0	7.6	2.81	2.68	0	3.0	0.76	0.65	0	10.5	2.56	3.41	−3.3	−0.8	−1.66	0.61	3.5	11.7	6.30	1.82
V_L	0	1.5	0.21	0.42	0	7.0	1.13	1.34	0	7.0	2.00	1.60	−1.0	1.0	0.05	0.41	0.5	8.5	3.27	1.85
V_F	0	1.2	0.29	0.37	0	13.0	6.68	2.81	0	6.5	0.80	1.28	0.2	2.8	1.46	0.62	4.0	13.0	7.66	2.54
V_1	0	0	0	0	1.0	9.6	4.16	2.33	3.4	24.0	11.05	5.03	−4.0	5.6	1.23	1.88	6.6	26.8	15.21	5.98
V_2	0	0	0	0	4.0	20.8	9.05	3.68	3.0	38.8	16.23	7.30	2.4	11.0	6.22	1.90	15.0	46.0	25.27	7.47
V_3	0	0.4	0.013	0.072	6.0	54.6	16.70	9.78	0	22.0	9.05	5.62	3.6	12.0	6.26	1.89	12.6	54.6	25.75	8.37
V_4	0	3.0	0.37	0.68	12.2	46.0	22.31	7.10	0	16.0	5.32	4.13	2.4	11.0	5.66	1.84	18.0	51.6	27.63	6.71
V_5	0	3.4	0.57	0.91	8.8	33.0	18.78	6.91	0	9.6	1.93	2.22	2.0	9.6	4.59	1.82	11.2	33.2	20.70	6.50
V_E	0	0	0	0	2.0	12.8	5.81	2.49	0	16.2	6.09	4.78	0.2	5.2	2.55	1.11	5.6	24.2	11.91	4.33

extremity leads V_R, V_L and V_F found by Kossmann and Johnston (11) in their study of thirty normal subjects are given in Table I, which is reproduced in modified form from their paper.

Unipolar limb leads. Unipolar leads from the right arm (V_R), left arm (V_L) and left leg (V_F) furnish a valuable means of comparing the potential variations of these extremities with those of the right and left sides of the precordium, and consequently of establishing relations between the complexes of the standard limb leads and those of the precordial leads. Unipolar limb leads may be taken by placing the exploring electrode used in taking the precordial leads on each of the extremities in turn or by transferring the wire connected to this electrode to each of the extremity electrodes. When this is done the deflections obtained are often inconveniently small unless the sensitivity of the electrocardiograph is increased to once and a half or twice its normal value. Goldberger (12) has introduced a procedure which avoids this difficulty. By breaking the connection between the central terminal and the extremity upon which the exploring electrode has been placed he increases the size of the deflections obtained by fifty per cent without changing the sensitivity of his apparatus. The standard limb leads and the unipolar limb leads are, of course, directly and simply related. The deflections of any two leads of either set furnish all the data needed to compute the deflections of the other four. The equations which express these relations have been published elsewhere (5) and need not be repeated here. It may, however, be pointed out here that when the ventricular complexes of leads I and II are similar in form the complexes of lead V_R resemble the complexes of these leads turned upside down; that when the complexes of leads III are the inverse of those of lead I, the complexes of lead V_L are like those of lead I; and that when the complexes of leads II and III are alike those of lead V_F have the same form. It should also be remembered that at any instant the algebraic sum of the potentials of the three extremities is equal to zero.

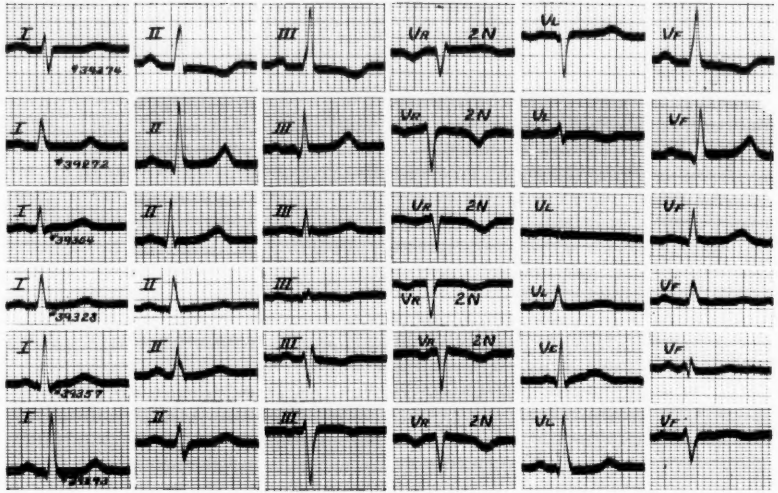


Fig. 3

The bipolar (leads *I*, *II*, and *III*) and unipolar (leads V_R, V_L and V_F) electrocardiograms of six subjects arranged according to the position of the mean electrical axis of *QRS*. The unipolar limb leads were taken with the galvanometer at twice the normal sensitivity (2N). Assuming that the *QRS* deflections of the precordial leads, had they been taken, would have been of the normal type, the heart was in the vertical position in the first, in the semivertical position in the second and third, in the intermediate position in the fourth, in the semihorizontal position in the fifth, and in the horizontal position in the sixth case. This figure appeared in an article by Wilson, Johnston, Cotrim and Rosenbaum (13).

The position of the heart from the electrocardiographic standpoint. The standard and unipolar electrocardiograms of six subjects are reproduced in Fig. 3. Not all of these tracings are normal but so far as the *QRS* complexes are concerned similar curves could be selected from any collection of normal records. They are arranged in accordance with the position of the mean electrical axis of *QRS*; the one which displays the greatest degree of right axis deviation is at the top and the one which displays the greatest degree of left axis deviation at the bottom. It will be noted that the potential variations of the right arm (V_R) were always of the same general kind. In some cases this extremity was slightly positive at the beginning, at the end, or both at the beginning and at the end of the *QRS* interval, but throughout by far the greater part of this interval it was conspicuously negative in all. The potential variations of the other two extremities varied greatly with the position of the electrical axis. When this axis was shifted to the right as in the first electrocardiogram the potential variations of the left arm (V_L) were like those which occur over the anterior surface of the dog's right ventricle and over the right side of the precordium in normal subjects. The potential variations of the left leg, (V_F) on the other hand, resembled those which occur over the anterolateral surface of the dog's left ventricle and over the left side of the precordium in normal subjects. When the electrical axis was shifted to the left as in the last electrocardiogram just the reverse was the case; the potential variations of the left arm resembled those normally found on the left side of the precordium, and the potential variations of the left leg those normally found on the right side (13).

When the heart is normal the precordial electrocardiogram has essentially the same form regardless of whether the limb leads display right axis deviation, no axis deviation, or left axis deviation. It is evident, therefore, that in normal subjects with right axis deviation the potential variations of the right ventricular surface are transmitted to the left arm and the potential variations of the left ventricular surface to the

left leg, whereas in normal subjects with left axis deviation the potential variations of the right ventricular surface are transmitted to the left leg and the potential variations of the left ventricular surface to the left arm. These differences in the magnitudes of the contributions made by the right and left ventricular surfaces to the potential variations of arm and leg can hardly depend upon factors lying within the heart itself; they must be due to variations in the spatial relations between the surfaces of the two ventricles and the attachments of the extremities to the trunk. When the electrocardiogram is normal there is an obvious correlation between the inclination of the mean electrical axis of *QRS* and the position of the heart. When the long axis of the heart is nearly vertical right axis deviation is usually present and when the long axis of the heart is nearly horizontal left axis deviation is usually present. We are, therefore, justified in using the terms "vertical" and "horizontal" in defining the position of the heart from the purely electrocardiographic standpoint. So far as the normal heart is concerned the first five of the six cardiac positions described below could be defined equally well in terms of the position of the electrical axis, but if that were done the definitions could not be applied when the heart was abnormal.

From the purely electrocardiographic standpoint we may then define the following six positions of the heart (see Fig. 3).

Vertical position:

(a) The ventricular complexes of lead V_L resemble those of leads V_1 and V_2.

(b) The ventricular complexes of lead V_F resemble those of leads V_5 and V_6.

Semivertical position:

(a) The ventricular complexes of lead V_F resemble the ventricular complexes of leads V_5 and V_6.

(b) The *QRS* deflections of lead V_L are small.

Intermediate position:

(a) The ventricular complexes of leads V_L and V_F are similar in form and size and like those of leads V_5 and V_6.

Semihorizontal position:

(a) The ventricular complexes of lead V_L resemble those of leads V_5 and V_6.

(b) The *QRS* deflections of lead V_F are small.

Horizontal position:

(a) The ventricular complexes of lead V_L resemble those of leads V_5 and V_6.

(b) The ventricular complexes of lead V_F resemble those of leads V_1 and V_2.

Indeterminate position:

There is no obvious relationship between the ventricular complexes of the limb leads and those of the precordial leads.

As long ago as 1910 Kraus and Nicolai (13) asserted that the deflections obtained by leading from the surface of the body are very similar to those that would be obtained by leading from those parts of the heart's surface beneath and nearest the electrodes. They based this opinion upon the principles that govern the distribution of electric currents in volume conductors. As regards precordial leads we reached a similar conclusion long before we knew that others had held this view, and we have demonstrated that there is a very close relation between the potential variations of a precordial electrode and the potential variations of the underlying ventricular surface. There is considerable evidence of a less direct kind that the potential variations of the right arm are similar to the potential variations of those parts of the heart's surface that are nearest the right shoulder; that the potential variations of the left arm resemble the potential variations of those parts of the heart's surface that are nearest

the left shoulder; and that the potential variations of the left leg are like the potential variations of the heart's diaphragmatic surface.

I have already mentioned that the potential of the right arm is ordinarily negative throughout the greater part of the *QRS* interval. The reason lies in the relation of the right shoulder to the great valvular orifices at the base of the heart. The negativity of the ventricular cavities is transmitted through these orifices to the adjacent parts of the body including the right shoulder and the right arm. The potential variations of this extremity do not, of course, represent the potential variations of the ventricular cavities in pure form, but mixed with the potential variations of other regions and particularly with those of the surface of the right ventricle. Over the thinner parts of the free wall of this chamber the potential is normally weakly positive at the very beginning of the *QRS* interval but during the rest of this interval it does not differ from that of the ventricular cavity. It is, therefore, frequently impossible to say whether the base of the heart or the right ventricular surface contributed most to the potential variations of the right arm. The same is sometimes true with reference to the potential variations of the left arm.

We also know that when the heart assumes a more vertical position or turns about its long axis in the direction required to bring the left ventricle below the right the potential variations of the left leg become more like those of the left ventricular surface and less like those of the right ventricular surface while the potential variations of the left arm change in the opposite way. When the heart assumes a more horizontal position or twists about its long axis in the direction required to bring the right ventricle below the left the effects upon the potential variations of the left arm and left leg are the reverse of those described.

It is also true that in myocardial infarction the potential variations that occur at the epicardial surface of the infarct are transmitted to the left arm when the anterolateral wall

of the left ventricle is involved and to the left leg when the diaphragmatic wall is involved.

But although the anatomical position of the heart and its electrocardiographic position are very closely related there are reasons for believing that the correlation between the two cannot be perfect. It is evident that those parts of the right ventricular surface which play the most important role in determining the potential variations of the right side of the precordium are not identical with those parts of this surface which play the most important role in determining the potential variations of the left arm or left leg, as the case may be. A similar statement may be made with reference to those parts of the left ventricular surface which contribute most to the potential variations of the left side of the precordium. Significant relations between the ventricular complexes of the precordial leads and the ventricular complexes of the unipolar limb leads of the kind upon which the determination of the electrocardiographic position of the heart is based can, therefore, be expected to occur only when potential variations of one kind take place simultaneously over a large part of the right ventricular surface and potential variations of another kind take place simultaneously over a large part of the left ventricular surface. This is likely to happen when the heart is normal, in right and in left bundle branch block, and in preponderant hypertrophy of the right or left ventricle. It is much less likely to happen when there are lesions which give rise to local modifications of the potential variations at the surface of the heart or have opposite effects upon the potential variations of different parts of the surface of the same ventricle. For this reason and because the potential variations of the extremities often represent complicated mixtures it is often impossible in cases of myocardial disease to establish any relation between the potential variations of the precordium and the potential variations of the extremities.

It should be noted that the electrocardiographic position of the heart may change even though the anatomical position

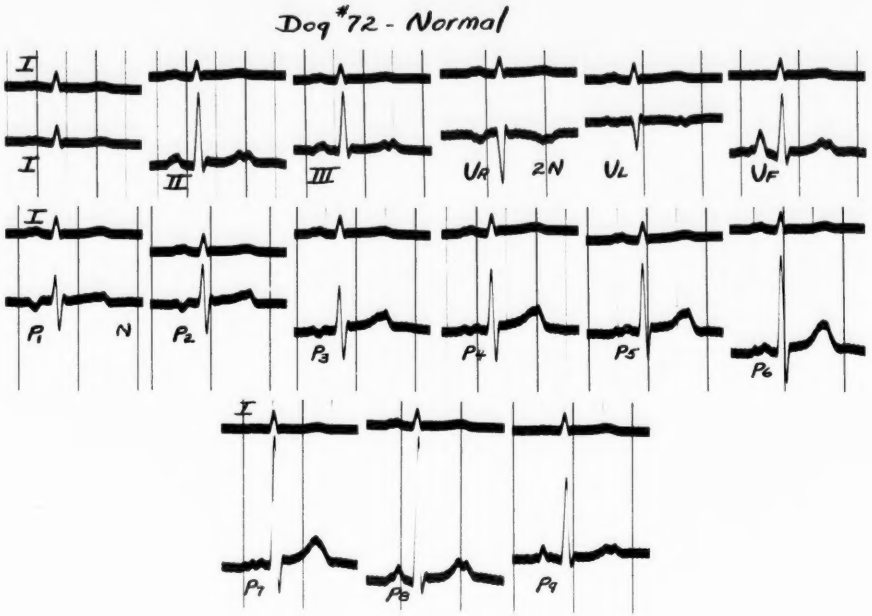


Fig. 4
The standard electrocardiograms, unipolar electrocardiograms, and precordial electrocardiograms of a normal dog. The potential variations of the left hind leg (V_F) were similar to those of the left side of the precordium (P_7, P_8, and P_9).

remains the same. This is true because the former is determined not only by the orientation of the surfaces of the two ventricles, but also by the distribution of potential variations of various kinds on each of them. In bundle branch block, for example, the regions in which the same kind of potential variations occur at the same time are not exactly the same in extent and configuration as when the heart is normal. Consequently the onset of bundle branch block may, although it usually does not, alter the position of the heart from the electrocardiographic standpoint.

The precordial electrocardiograms of dog and man. The electrocardiograms reproduced in Fig. 4 represent observations made in the course of an experiment upon a normal dog. Standard limb leads, unipolar limb leads and nine unipolar precordial leads were taken. The first precordial lead was from a point *4 cm.* and the second from a point *2 cm.* to the right of the midline; the other precordial leads were taken by moving the exploring electrode across the precordium in a base-apex direction and were from points *2 cm.* apart. The precordial curves closely resemble the normal human curves obtained by the same method. The potential variations of the two forelegs were similar; both extremities were negative throughout the greater part of the *QRS* interval (leads V_R and V_L). The ventricular complexes of lead V_F are like those obtained from the left side of the precordium (leads P_5, P_6, P_7, P_8 and P_9) and the deflections of lead V_L are smaller than those of the other unipolar limb leads. The heart was, then, in the semivertical position.

The electrocardiograms shown in Fig. 5 are reproduced to demonstrate that there are no essential differences between the precordial electrocardiograms obtained in canine bundle branch block and those obtained in human bundle branch block. The curves of the first set represent left branch block in man; those of the second set left branch block in the dog. In both cases the ventricular complexes of the leads from the right side of the precordium display small *R* and broad

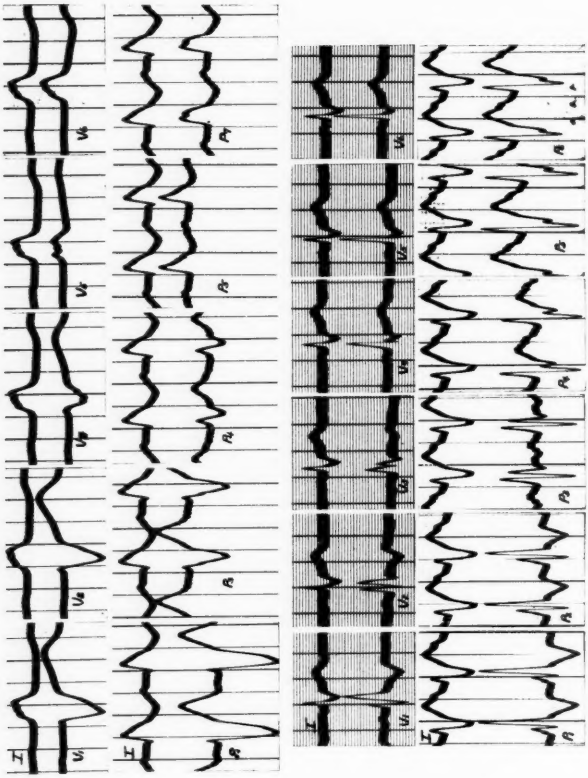


Fig. 5
The precordial electrocardiogram in bundle branch block. (1) Human left branch block. (2) Canine left branch block. (3) Human right branch block. (4) Canine right branch block.

S deflections indicating that the excitatory process arrived at the surface of the right ventricle very early in the *QRS* interval. The ventricular complexes of the leads from the left side of the precordium, on the other hand, are dominated by broad notched *R* waves of the kind seen in direct leads from the anterolateral wall of the left ventricle after section of the left branch of the His-bundle. The curves of the third set represent human right branch block and those of the fourth set canine right branch block. Here the complexes of the leads from the right side of the precordium display a small initial *R* and a tall final *R'* deflection and are similar to those obtained by leading directly from the surface of the right ventricle after the right branch of the His-bundle has been cut. The ventricular complexes of the leads from the left side of the precordium show narrow *R* and broad notched *S* deflections indicating early activation of the left ventricular surface. It is evident that if precordial leads, instead of limb leads, had been used in the early studies of experimental and clinical bundle branch block, human left branch block could never have been mistaken for right branch block over a period of nearly twenty years. The confusion which prevailed over this long period arose because the dog's heart is almost always in the semivertical position whereas in patients with arteriosclerotic heart disease the heart is usually in either the semihorizontal or the horizontal position. Since the relations between the surfaces of the two ventricles and the attachments of the extremities to the trunk are not constant in man the differentiation of human right from human left bundle branch block, on the basis of the data furnished by limb leads alone, is sometimes impossible.

Comparison of precordial and direct leads. The similarities and differences between unipolar precordial leads and unipolar direct leads from the anterior surface of the heart can be appreciated only when these two kinds of leads are directly compared. In a number of experiments on dogs we cut one of the branches of the His-bundle under aseptic conditions, restored the chest and allowed the animal to recover com-

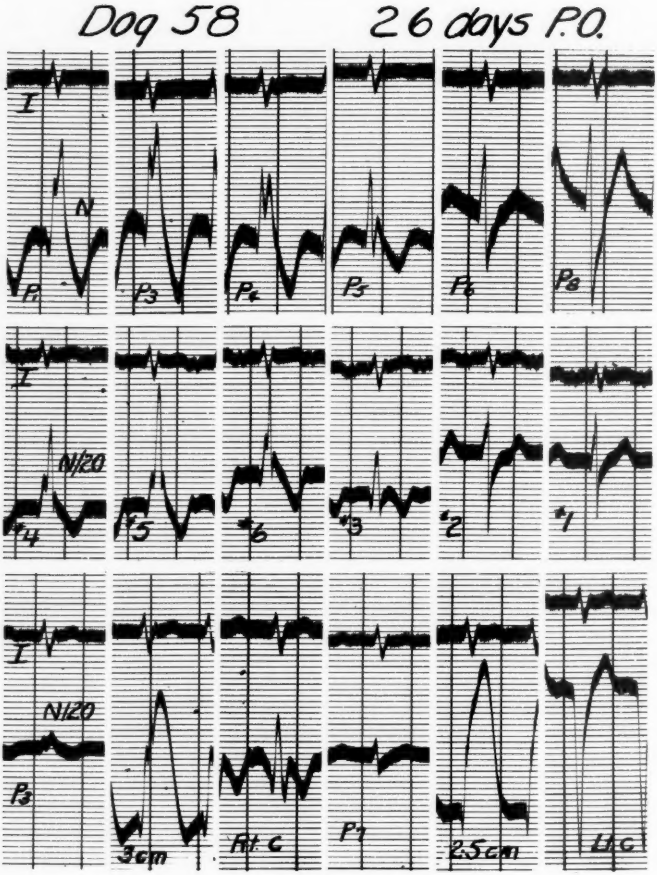


Fig. 6

Precordial and direct leads in canine right branch block. (1) The precordial electrocardiogram. (2) Direct leads from three points on the right and three points on the left ventricle. (3) *Rt. C*: Potential variations of the cavity of the right ventricle. *Lt. C*: Potential variations of the cavity of the left ventricle.

pletely. Approximately one month after this operation unipolar precordial leads were taken with the chest intact. The heart was then exposed by splitting the sternum and the potential variations of numerous points on its anterior surface were recorded.

The curves shown in Fig. 6 are from an experiment in which the right branch of the His-bundle was cut 26 days before the electrocardiographic studies were made. Six of the precordial electrocardiograms are reproduced (P_1, P_3, P_4, P_5, P_6 and P_8). Below these are the records of the potential variations of three points on the right ventricle (points *4*, *5* and *6*), one point near the interventricular sulcus (point *3*) and two points on the surface of the left ventricle (points *1* and *2*). Point *4* was on the conus, point *5* near the atrioventricular groove, point *6* in the central region, and points *2* and 1 on the left border above the apex.

The first three curves of the bottom row were obtained before the chest was opened by thrusting a sharp exploring electrode, insulated except at the extreme tip, through the chest wall on the right side of the precordium. The first curve was taken when the tip of the electrode was just beneath the skin, the second when it touched the heart and yielded a monophasic response, and the third when it was in the cavity of the right ventricle. The last three curves were obtained in the same way except that the sharp electrode was thrust through the chest wall at a point just inside the apex beat and consequently entered the cavity of the left ventricle. It will be noted that the cavity of this chamber was negative throughout the *QRS* interval. The potential variations of the cavity of the right ventricle were not of the usual form inasmuch as the positivity of this chamber due to the spread of the impulse through the septum from left to right did not occur at the very beginning of the *QRS* interval as it ordinarily does. The *R* wave to which it gave rise is, therefore, preceded by a *Q* deflection.

The curves shown in Fig. 7 are from an experiment in which the left branch of the His-bundle was cut 27 days

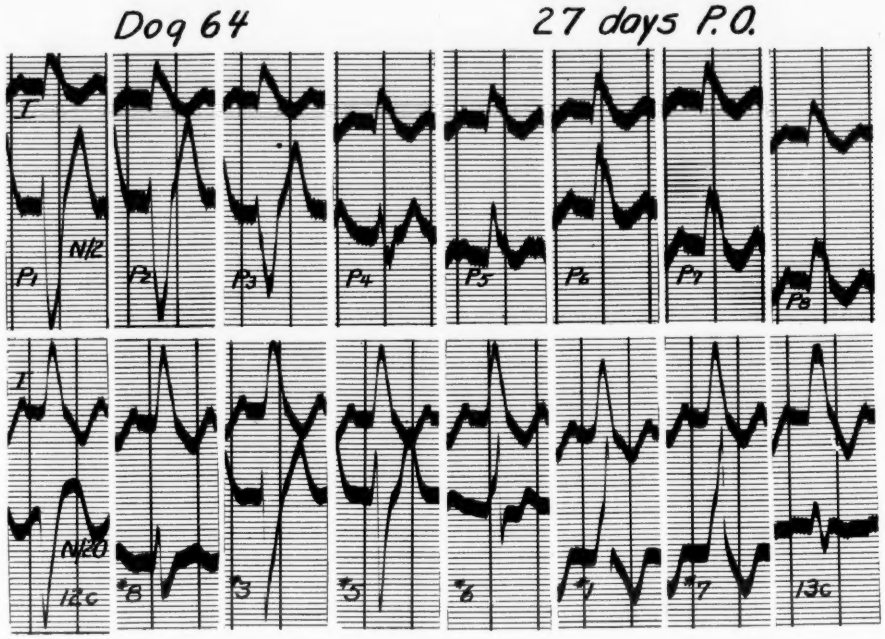


Fig. 7

Precordial and direct leads in canine left branch block. (1) The precordial electrocardiogram. (2) The potential variations of three points (*8, 3, 5*) on the right and three points (*6, 1, 7*) on the left ventricle. Potential variations of the cavity of the right (*12c*) and of the cavity of the left (*13c*) ventricle.

before the electrocardiographic study was made. Eight precordial electrocardiograms are reproduced. Below these are records of the potential variations of the cavity of the right ventricle (*12c*), three points on the right ventricle (*8,3,5*), three points on the left ventricle (*6,1,7*), and the cavity of the left ventricle (*13c*). Point *8* was near the atrioventricular groove, point *3* in the upper part of the central region, point *5* close to the trabeculated region, point *6* near the apex, and points *1* and *7* on the left border well above the apex. It will be noted that the cavity of the right ventricle was negative throughout the *QRS* interval, while that of the left ventricle displayed initial positivity.

The similarity between the potential variations of the exposed surface of the right ventricle and the potential variations of the right side of the precordium, and between the potential variations of the surface of the left ventricle and those of the left side of the precordium in these two experiments requires no comment. We may, however, point out that the first peak of the bifid *R* waves often recorded over the delayed ventricle occurs when the cavity of that ventricle is positive, whereas the second peak, which marks the beginning of the intrinsic deflection, occurs when the cavity is negative. In those cases in which the peak of this broad *R* wave is not divided the first peak is usually represented by a shoulder on its ascending limb.

Human bundle branch block. There can be no reasonable doubt that in man as in the dog the potential variations of the right side of the precordium (leads V_1 and V_2) ordinarily resemble the potential variations of the anterior surface of the right ventricle while the potential variations of the left side of the precordium (leads V_5 and V_6) ordinarily resemble the potential variations of the anterolateral surface of the left ventricle.

The electrocardiograms shown in Fig. 8 are those of a man who was examined both before and after he developed left bundle branch block. The tracings of March 28, 1940, display slight left axis deviation and flat *T* waves in lead II;

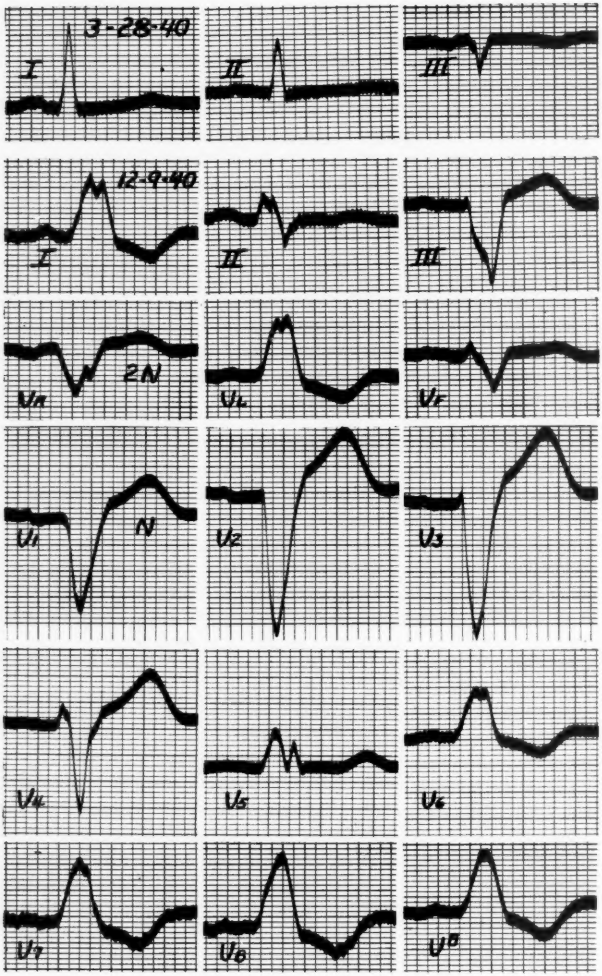
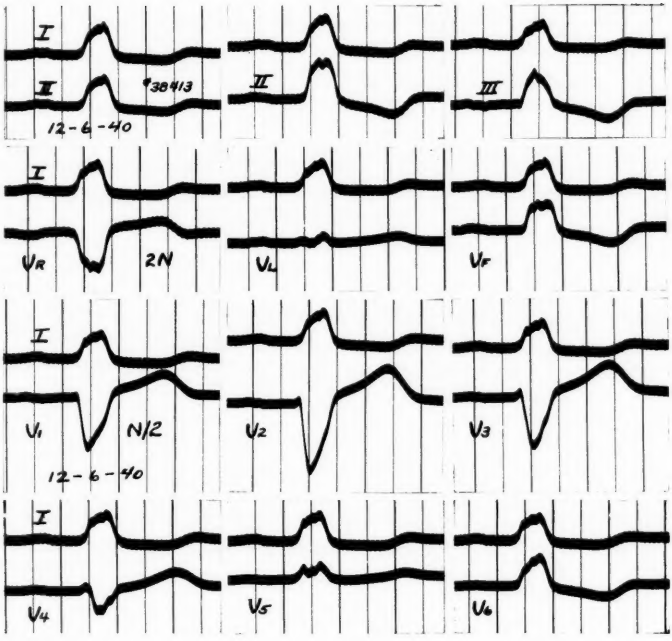


Fig. 8

Left bundle branch block; heart in the horizontal position.



Left bundle branch block; heart in the semivertical position.

the *QRS* interval measures about 0.09 second. On December 9, 1940, left branch block was present. On this occasion unipolar limb leads, unipolar precordial leads, and unipolar leads from the left infrascapular region were taken. The potential variations of the left arm (V_L) were similar to those of the left side of the precordium and the left infrascapular region, whereas the potential variations of the left leg were like those of the right side of the precordium in general outline. The heart was then in the horizontal position.

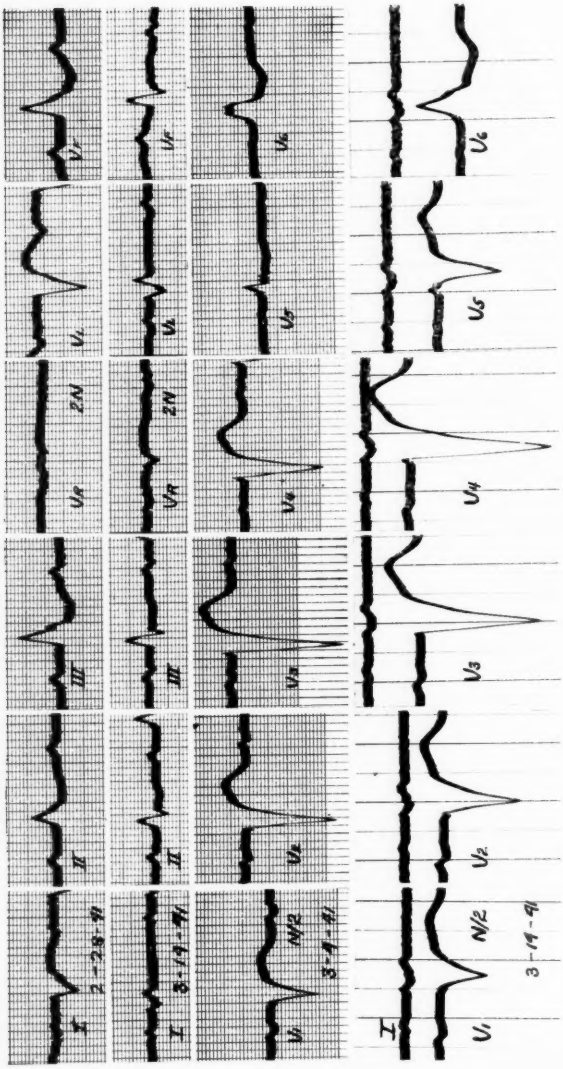


Fig. 10
Left bundle branch block; heart in the vertical position. (From an article by Wilson (15).)

The electrocardiograms shown in Fig. 9 are those of a tall thin man who was examined for the first time on February 3, 1937, when his electrocardiogram was normal. There was no deviation of the mean electrical axis at this time. On April 14, 1940 left bundle branch block was present and there were broad notched *R* deflections in leads I and II and broad *S* deflections in lead III. The precordial electrocardiograms of that date differ in no essential particular from those reproduced and the heart was evidently in the semihorizontal position. On November 27, 1940 and on December 6, 1940 the ventricular complexes of the standard limb leads were of the concordant type. The unipolar leads taken on the latter date show that the potential variations of the left leg were like those of the left side of the precordium and the left infrascapular region. The potential variations of the left arm were small on both occasions. On these two days the heart was in the semivertical position.

The patient whose electrocardiograms were reproduced in Fig. 10 was under observation from February 26, 1941, until March 22, 1941. The ventricular complexes of the standard limb leads were quite variable in form. The curves taken on February 28 display discordant ventricular complexes of the kind ascribed to bundle branch block of the rare type. Those taken on March 14 show small bizarre *QRS* deflections in lead I and diphasic QRS complexes with broad initial *R* waves in leads II and III. In the tracings taken on February 26, March 13, March 18 and March 26 the ventricular complexes are intermediate in form between the two types reproduced. Precordial electrocardiograms were taken on March 4 and on March 14 and the two sets of curves differ only as regards the form of the ventricular complexes of lead V_5. Differences of this kind have no important significance. They are apparently due to variations in the position of the heart or to slight variations in the locations of the precordial points explored. The precordial electrocardiograms demonstrate beyond question that the conduction defect was in the left branch of the His-bundle and not in the right (*15*). When they are compared

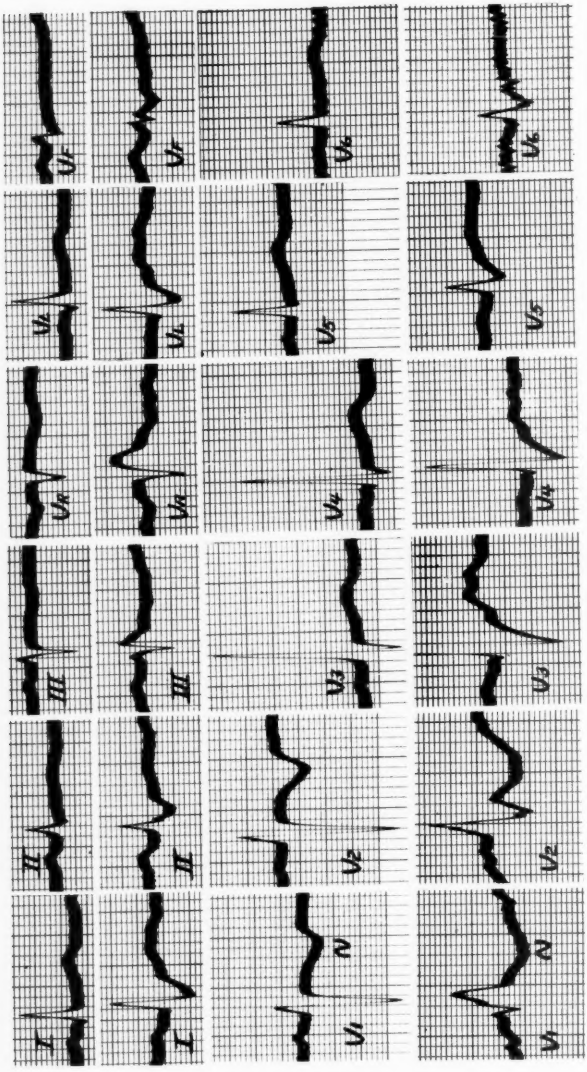


Fig. 11
Right bundle branch block induced by quinidine; heart in the semihorizontal position.

with the unipolar extremity curves taken on February 28, it is clear that the heart was then in the vertical position. On that date the potential variations of the left arm were like those subsequently found on the right side of the precordium and the potential variations of the left leg like those found on the left side of the precordium. On March 14 the heart was in a somewhat different position. On that day the potential variations of the left arm and of the left leg were apparently complicated mixtures of components transmitted from the surface of the right ventricle and components derived from the surface of the left ventricle.

The electrocardiograms reproduced in Fig. 11 are those of a young woman with ulcerative colitis who developed right bundle branch block on two different occasions after the administration of 0.4 gm. of quinidine sulphate. Before the drug was given slight left axis deviation was present, but apart from inversion of the T waves in lead V_2 both the standard and the precordial electrocardiogram were within normal limits. In both sets of curves the ventricular complexes of lead V_L resemble those of leads V_5 and V_6 and the deflections of lead V_F are small. The heart was then in the semihorizontal position. It will be noted that the onset of right branch block produced more striking changes in the form of the ventricular complexes of lead V_1 than in the form of those of leads V_5 and V_6. In the latter the R wave was slightly smaller and the S deflection deeper and broader after the block than before; in the former the deep S originally present was replaced by a broad slurred R' when the block developed. When the cardiac mechanism was normal the transitional zone which yields complexes intermediate in form between those obtained from points farther to the left and those obtained from points farther to the right lay between precordial points two and three. When right branch block was present it lay between points one and two; only the first precordial lead yielded ventricular complexes of the kind seen in direct leads from the surface of the right ventricle. On the second occasion

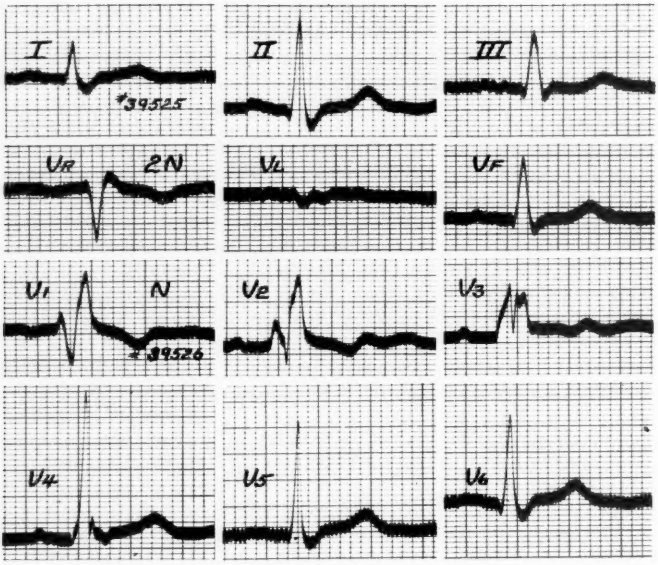


Fig. 12
Right bundle branch block; heart in the semivertical position.

on which right branch block was induced by quinidine this apparent shift in the transitional zone did not occur.

The electrocardiograms reproduced in Fig. 12 represent right bundle branch block in a man whose heart was in the semivertical position. The precordial electrocardiogram displays features usually associated with this conduction defect; the transitional zone lay between points three and four. In the standard limb leads the ventricular complexes are of the concordant type. The potential variations of the left leg were like those of the left side of the precordium and the potential variations of the left arm were very small.

The electrocardiograms reproduced in Fig. 13 are those of a man, aged sixty-two years, with arteriosclerotic heart disease. The ventricular complexes of the standard limb leads suggest that left branch block was present. It should be

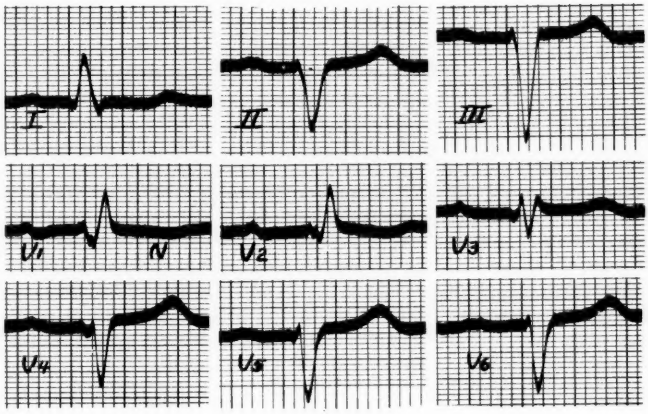


Fig. 13
Right bundle branch block; heart in the vertical position. (From an article by Wilson (15).)

noted, however, that a Q and an S deflection are present in lead I; both of these components are extremely rare in this lead in left branch block and common in right. The precordial electrocardiogram shows that the conduction defect was on the right side, but it is peculiar as regards the size of the R deflection in leads V_5 and V_6. The small size of the R waves in these leads suggests that the block was complicated by some factor which diminished the magnitude of the forces produced by activation of the anterolateral wall of the left ventricle. Although no unipolar limb leads were taken the form of the complexes of the standard limb leads shows that the potential variations of the left leg were like those of the left side of the precordium and the potential variations of the left arm like those of the right side of the precordium. The heart was, therefore, in the vertical position.

When the QRS interval measures 0.12 second or more and the QRS complex in lead I is monophasic and consists of a broad, slurred, flat-topped, or bifid R deflection the precordial curves are, almost always, characteristic of left branch block. When the QRS interval measures 0.12 second or more and the QRS complex

in lead I is biphasic or triphasic and ends with a broad, slurred or notched *S* deflection the precordial curves are, in the vast majority of cases, characteristic of right bundle branch block. The occasional exceptions to these general rules are due to a number of different causes. When the heart is in the vertical position the limb leads may suggest that right branch block is present when the precordial curves are characteristic of left branch block and vice versa. In other instances the prolongation of the *QRS* interval is due to a general depression of the conductivity of the Purkinje tissue or to local lesions involving the subendocardial Purkinje network over a considerable area ("arborization block") (*16*). The latter is seen most often in association with extensive myocardial infarction.

When the *QRS* interval measures 0.12 second or more, and the precordial electrocardiogram shows some but not all of the features typical of right or of left branch block the conduction defect cannot be located with certainty. Sometimes this situation arises because this defect is associated with lesions which modify its effects upon the electrocardiogram. Sometimes it depends upon peculiarities in the extent or location of the transitional zone, which separates the parts of the precordium that display potential variations like those of the anterior surface of the right ventricle from the parts of the precordium that display potential variations like those of the anterolateral surface of the left ventricle. This may be so far to the left or so far to the right that all of the six standard precordial leads yield ventricular complexes of the same type. When this happens it is desirable to take additional unipolar leads so arranged as to cross this zone. It is still impossible to say why the transitional zone is so variable in width and location. Finally, it should be emphasized that in many cases of bundle branch block there is no obvious relation between the form of the ventricular complexes of the precordial leads and the form of the ventricular complexes of the limb leads. In these cases the position of the heart must be classed as indeterminate.

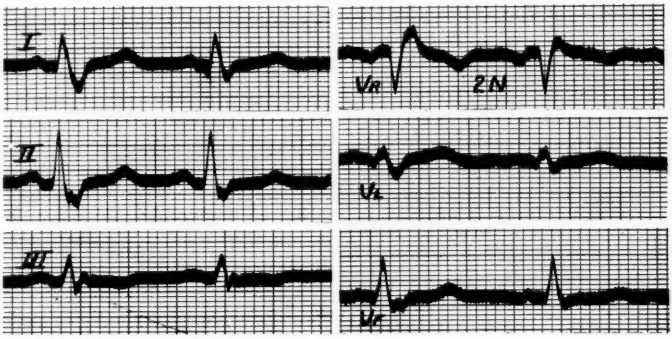


Fig. 14

Complete and incomplete right bundle branch block; standard and unipolar limb leads.

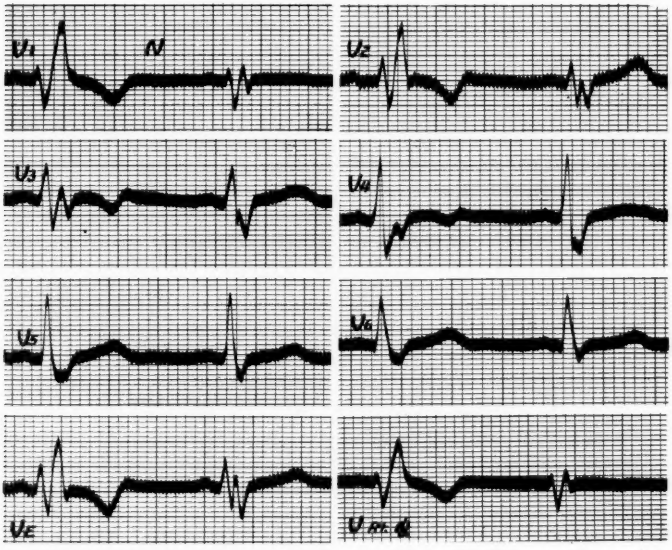


Fig. 15

Complete and incomplete right branch block; precordial leads.

When the *QRS* interval is abnormally long but measures distinctly less than 0.12 second the precordial curves are seldom characteristic of complete bundle branch block. In rare instances they are typical of right branch block even though the *QRS* interval of the limb leads is not much greater than 0.10. In most of the cases of this kind that we have seen we suspected that the right ventricle was greatly hypertrophied and it is possible that this condition was in part responsible for the form of the precordial electrocardiogram.

Unipolar precordial leads make it possible to diagnose incomplete right bundle branch block in many cases in which it cannot be recognized by other means. Except under special circumstances the diagnosis of incomplete left branch block is still very difficult to make with certainty.

The electrocardiograms reproduced in Figs. 14 and 15 represent the alternation of complete with incomplete right branch block. The first ventricular complex of each pair represents complete and the second incomplete block. It will be noted that as regards their earliest components the paired complexes are identical in all leads. In the case of the limb leads and the leads from the left side of the precordium (V_5 and V_6) the sole difference lies in the width of the final *QRS* component and the size of the *T* deflection. In the leads from the right side of the precordium, (V_1 and V_2) from the tip of the ensiform process (V_E) and from the right nipple line the difference is much greater; the large final R' deflection of the first complex is very much smaller (V_1 and V_E) or embryonic* in the second (V_2).

The presence of such small or embryonic R' deflections in addition to an initial *R* wave in the leads from the right side of the precordium strongly suggests that incomplete right branch block is present particularly when the *QRS* interval is slightly increased and there is a broad *S* wave in lead I and in the leads from the

*This term is used to indicate that the peak of this deflection does not rise above the level of the trace at the beginning of the *QRS* interval.

left side of the precordium. It is advisable to take precordial leads whenever the *QRS* complex of lead I is of this kind.

In incomplete left bundle branch block the precordial electrocardiogram resembles that seen in preponderant hypertrophy of the left ventricle and the two conditions frequently co-exist. The presence of a *Q* deflection in the leads from the left side of the precordium excludes the former, but the absence of this deflection does not differentiate one from the other.

Transient Branch Block. In the vast majority of the cases of human bundle branch block there is no way of knowing whether all the electrocardiographic abnormalities present are due to the conduction defect or whether some of them are the result of the other lesions which are almost always present. Cases in which the electrocardiogram is normal after branch block disappears or immediately before it develops are, therefore, of unusual interest. In such cases the effects produced by the block are uncomplicated by other factors.

The electrocardiograms reproduced in Fig. 16 are those of a woman, aged 55 years, who had symptoms suggesting hyperthyroidism. She was under observation from October 13, 1936 until November 3, 1936. Left branch block was present when the patient was seen for the first time, but on October 19 the standard extremity curves were normal. At this time the precordial curves were normal as regards the *QRS* complexes, but the *T* waves were sharply inverted in leads V_1, V_2, V_3 and V_4. This case conforms to the rule that abnormalities which are confined to the leads from the right side of the precordium are not accompanied by similiar abnormalities in the limb leads. It illustrates the value of precordial leads in detecting lesions which produce electrocardiographic disturbances of this kind. On October 22 the branch block was again present. In the standard leads the broad notched *QRS* deflections were of low voltage and of the concordant type. In the precordial leads the voltage of the chief *QRS* deflection was so large that in

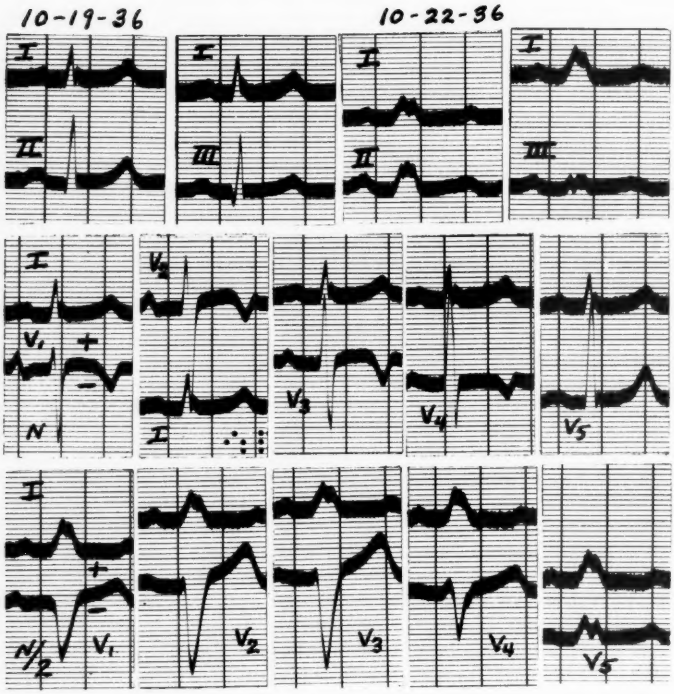


Fig. 16

Transient left branch block.

order to record it properly it was necessary to reduce the string galvanometer to one-half the normal sensitivity. It will be noted that the conduction defect greatly reduced the size of the R waves in the leads from the right side of the precordium, abolished the inversion of the T waves previously present in these leads, and shifted the transitional zone to the left. In the leads from the left side of the precordium (V_5 and V_6) it gave rise to monophasic QRS complexes consisting of broad, bifid R deflections. It is evident that the heart was in the semi-vertical position.

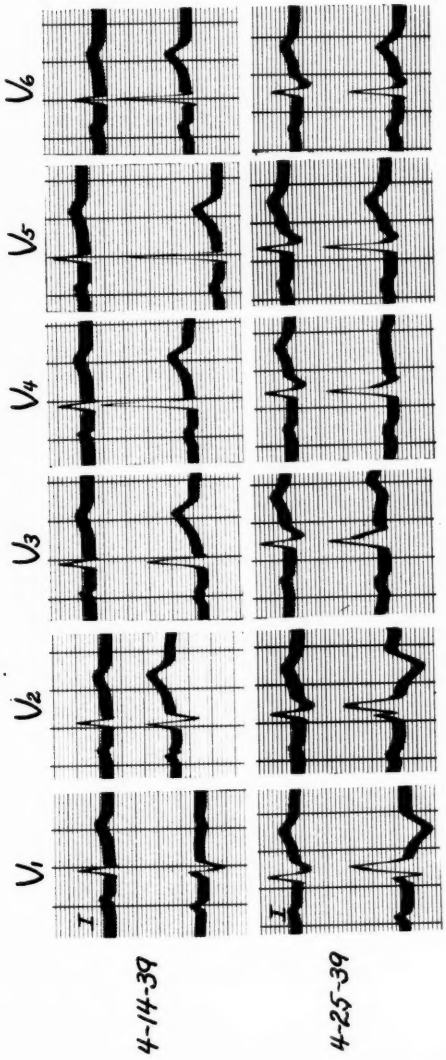


Fig. 17
Transient right branch block.

The electrocardiograms reproduced in Fig. 17 are those of a woman, aged 24 years, with rheumatic heart disease. She was under observation from November 16, 1938 until April 25, 1939. When she was first seen the electrocardiogram displayed rather large *P* deflections, but was not definitely outside normal limits. On April 5 the *P-R* interval measured 0.32 second and right branch block was present. On April 14 the electrocardiogram was normal and precordial leads were taken. On April 25 right branch block was again present and the precordial leads were repeated. It will be noted that the block gave rise to a large final *R'* deflection in leads V_1 and V_2 and inverted or flattened the *T* waves in the first four precordial leads, made the *R* deflections somewhat smaller in the leads from the left side of the precordium, and produced broad *S* waves in leads V_5 and V_6.

Ventricular hypertrophy. Preponderant hypertrophy of the left ventricle increases the thickness and the bulk of its walls. When uncomplicated its chief effect is, therefore, to exaggerate the normal difference in this respect between this chamber and its fellow. It is not then surprising that in left ventricular hypertrophy unipolar precordial leads yield curves which, in their broader aspects, are not unlike those obtained when the heart is normal. The chief differences are these: In left ventricular hypertrophy the voltage of the chief deflection of the *QRS* group is on the average much greater than normal, and the *QRS* interval is increased to 0.10 or even 0.11 second. In the leads from the right side of the precordium the *R* deflections are, on the average much smaller than normal and may be absent (*17*). The transitional zone is, as a rule, much displaced to the left. In the leads from the left side of the precordium *R*, and often *Q* as well, is abnormally large, the peak of *R* occurs abnormally late in the *QRS* interval, and the *T* deflections are inverted.

By increasing the bulk and thickness of its walls preponderant hypertrophy of the right ventricle tends to abolish or reverse the normal difference between it and the left. In

high grade right ventricular hypertrophy the precordial curves are, as regards their broader aspects, somewhat like those obtained in right branch block, but the *QRS* interval is not increased and the *QRS* deflections are not ordinarily broad, slurred, or notched. On the average the voltage of the chief *QRS* deflection is above normal although not as large as in left ventricular hypertrophy (*17*). In the leads from the right side of the precordium the *R* wave is very large, *Q* is frequently present, and *S* usually absent, and the *T* deflections commonly inverted. In some instances a small initial upward deflection precedes the first downward deflection of the *QRS* group, but we suspect that the presence of such a summit may be due to incomplete right branch block. In the leads from the left side of the precordium the *R* deflections are abnormally small and the *S* deflections abnormally large. In other words, the precordial curves are opposite in type to those obtained when the heart is normal.

In left ventricular hypertrophy the position of the heart has the same effect upon the mean electrical axis of *QRS* as in normal subjects. The electrocardiograms reproduced in Fig. 18 are those of a man, aged 54 years, with hypertensive heart disease. The standard extremity curves display left axis deviation, a *QRS* interval measuring approximately 0.10 second, and prominent *Q* waves, abnormally large *R* deflections, and inverted *T* waves in lead I. The precordial curves, which were taken with the galvanometer at one-half the normal sensitivity are characteristic of left ventricular hypertrophy. Although no unipolar limb leads were taken it is clear that the heart was in the horizontal or semihorizontal position.

The electrocardiograms reproduced in Fig. 19 are those of a woman aged 50 years with hypertensive heart disease. No axis deviation is present but the *R* deflections are obnormally tall and the *T* waves inverted in all of the standard limb leads. The *QRS* interval is slightly increased. The precordial curves, which were taken with the galvanometer

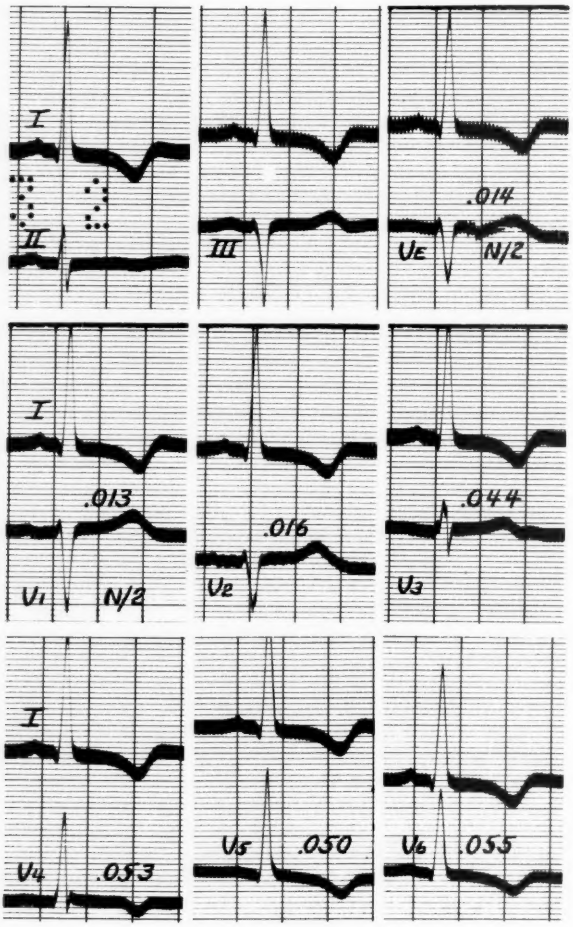


Fig. 18

Left ventricular hypertrophy; heart in the horizontal or semihorizontal position.

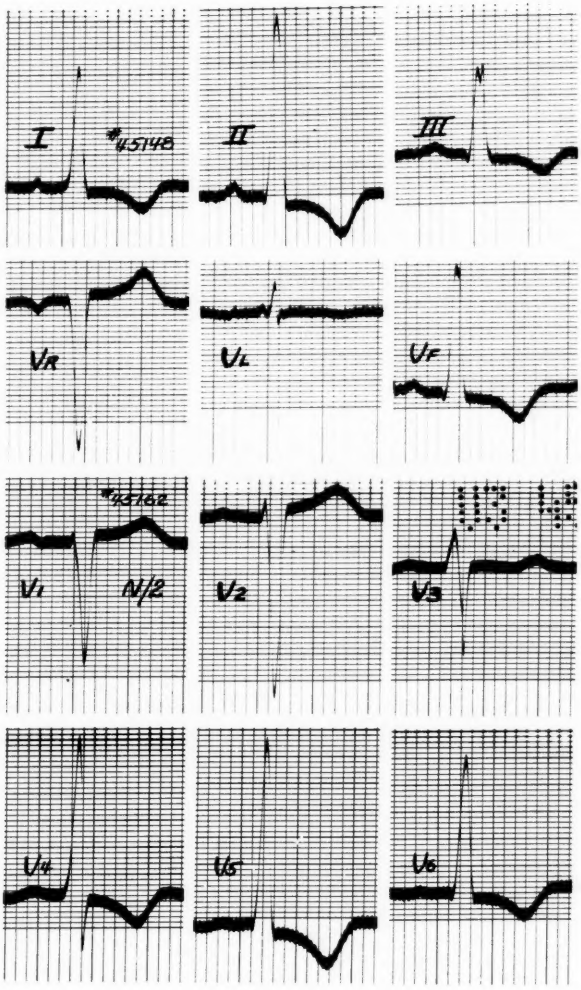


Fig. 19

Left ventricular hypertrophy; heart in the semivertical position.

at one-half the normal sensitivity, are characteristic of left ventricular hypertrophy. The potential variations of the left leg were like those of the left side of the precordium and the potential variations of the left arm were small. The heart was in the semivertical position.

The electrocardiograms reproduced in Fig. 20 are those of a young man, aged 16 years, with rheumatic heart disease, aortic stenosis and regurgitation and probably mitral stenosis, and an enormously enlarged heart. Right axis deviation is present, the *T* deflections are inverted in leads II and III, the *P-R* interval is increased, and the *P* deflections are large and deformed. Seven precordial leads were taken, the last, marked V_7, from the posterior axillary line at the level of the apex, which was not far inside this point. The precordial curves, which were taken with the galvanometer at one-half the normal sensitivity, are characteristic of left ventricular hypertrophy. Although unipolar limb leads were not taken it is evident the potential variations of the left leg were like those of the left side of the precordium and the potential variations of the left arm like those on the right side of the precordium. The heart was, therefore, in the vertical position.

The electrocardiograms of two patients with right ventricular hypertrophy are reproduced in Fig. 21. The curves of the upper set are those of a woman, aged 34 years, who had pulmonary hypertension of unknown origin. The limb leads display conspicuous right axis deviation. The precordial curves are characteristic of right ventricular hypertrophy; tall *R* waves, prominent *Q* waves, and inverted *T* waves are present in lead V_1 and small *R* and deep *S* waves in leads V_5 and V_6. The potential variations of the left arm were like the potential variations of the left side of the precordium, and the potential variations of the left leg resembled those of the right side. The heart was in the horizontal position. The curves of the second set are those of a man, aged 40 years, with mitral stenosis and aortic insufficiency. The precordial

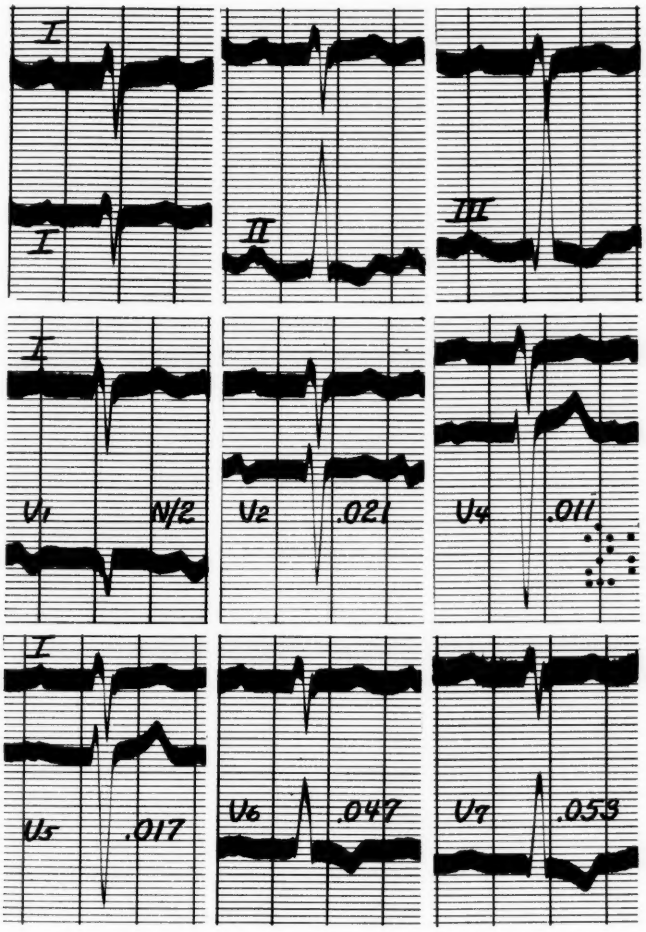


Fig. 20

Left ventricular hypertrophy; heart in the vertical position. (From an article by Wilson, Johnston, Cotrim and Rosenbaum (13).)

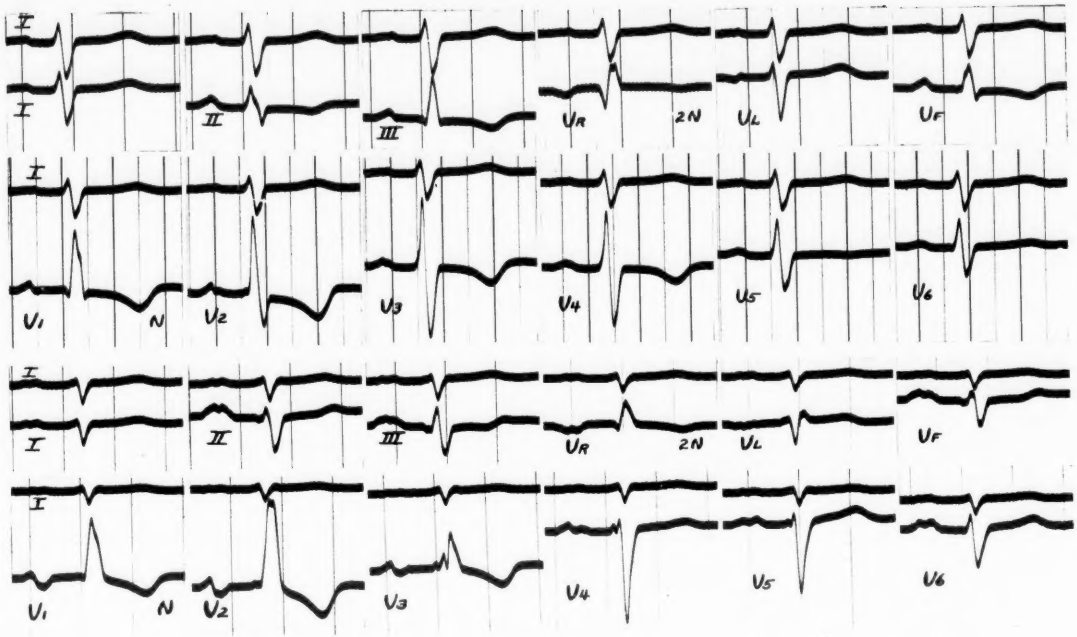


Fig. 21

Right ventricular hypertrophy. (1) Heart in the horizontal position. (2) Heart in the semivertical position.

curves are characteristic of right ventricular hypertrophy. Large S waves and small R waves are present in all of the standard limb leads. Here the ventricular complexes of the unipolar leg lead are like those of leads V_5 and V_6; the complexes of the right-arm lead are like those of lead V_1, and the complexes of the left-arm lead apparently represent a mixture of the potential variations of the other two extremities. In this case the heart was apparently in the semivertical position.

When the heart is greatly enlarged it is ordinarily in the horizontal or semihorizontal position. In either case left ventricular hypertrophy produces left, and right ventricular hypertrophy produces right axis deviation. When the heart is in the semivertical position left ventricular hypertrophy produces abnormally tall R waves and usually inverted T waves, and right ventricular hypertrophy produces large S waves in all of the standard limb leads. When the heart is in the vertical position left ventricular hypertrophy produces right, and right ventricular hypertrophy should produce left axis deviation although we have not as yet seen the last occur.

The precordial curves of left ventricular hypertrophy are distinguished from those of left bundle branch block by the absence of slurred or bifid R waves and the great frequency of Q deflections in the leads from the left side of the precordium. In left branch block the presence of Q in these leads suggests that the diagnosis is wrong or that septal lesions which prevent initial positivity of the left ventricular cavity are present. The QRS interval is almost always less than 0.12 second in hypertrophy, whereas in block it equals or exceeds this figure.

The precordial curves of right ventricular hypertrophy are distinguished from those of right bundle branch block by the absence of double or bifid R waves in the leads from the right side of the precordium, by the small size of the R waves in the leads from the left side of the precordium, and by the absence of a greatly increased QRS interval.

It must be admitted, however, that cases are frequently encountered in which it is impossible to say with certainty whether the electrocardiographic changes are due to hyper-

trophy alone or to hypertrophy plus incomplete bundle branch block.

Myocardial infarction. The character of the potential variations at the epicardial surface of an infarct depends upon the stage of infarction and upon whether the whole thickness of the ventricular wall or only the subendocardial layers of muscle are involved. Immediately after ligation of the anterior descending coronary artery or one of its branches in experiments upon the dog's heart direct leads from the region supplied by the vessel occluded display pronounced upward displacement of the *RS-T* junction which persists for many hours, but disappears when the muscle affected dies or recovers (18). This *RS-T* displacement is accompanied by a corresponding reduction in the voltage of the intrinsic deflection. When the whole thickness of the ventricular wall is involved and the infarcted muscle is already dead or incapable of responding to the excitatory impulse the potential variations of the part of the ventricular cavity adjacent to the infarct are faithfully transmitted to its epicardial surface. Since the ventricular cavities are normally negative throughout the *QRS* interval direct leads from the central part of the infarct then yield *QRS* complexes which consist of a monophasic downward deflection (*QS* deflection). In many cases some fraction of the muscle in this region remains alive and gives rise to an embryonic *R* wave which produces a notch on the descending or ascending limb of the single *QRS* component. When the subendocardial layers of muscle are for the most part dead or incapable of responding to the cardiac impulse and the outer layers of muscle are relatively normal the embryonic *R* rises above the base line and becomes a true *R* deflection. The *QRS* complex then consists of an abnormally large *Q* followed by a small *R* and often by an *S* deflection also. Since infarcts of the left ventricular wall are usually more extensive on the endocardial than on the epicardial side *QRS* complexes of this kind are commonly seen in direct leads from the marginal parts of the infarcted area (19). These same zones also yield deep, sharply inverted *T* deflections due to an increase in the duration

of the excited state at the epicardial surface. In the dog the *T* wave changes develop some hours after infarction begins and ordinarily disappear almost completely within twenty-four hours or less. In man they commonly last for some weeks or months.

The potential variations of the epicardial surface of the infarcted region are transmitted to the adjacent parts of the body. When the anterior wall of the left ventricle is involved they are transmitted to the precordium; if the anterolateral wall is involved they are also transmitted to the left arm. If the posterior wall is infarcted they are usually transmitted to the ventricular levels of the esophagus, to the back, and to the left leg. The potential variations of points at some distance from the heart, but on the same side of it as the infarcted region are, of course, a mixture of components derived from the epicardial surface of the infarct, from regions adjacent to it and from other parts of the ventricular surface. It is only when the infarct is large that characteristic curves are obtained by leading from regions distant from it. The potential variations of parts of the body on the side of the heart opposite to the one involved are, as regards their general character, the inverse of those that occur at the surface of the infarct.

The diagnosis of myocardial infarction can be made with certainty from the electrocardiogram alone only when characteristic changes in the *QRS* complex occur in association with *RS-T* displacement or typical changes in the *T* deflections. Changes in the *QRS* complex have more diagnostic value than displacement or changes in the *T* waves, when only electrocardiographic data are available, but they are not conclusive. When clinical data are available and are strongly suggestive of infarction and serial curves are taken, the opposite is true because of the relatively rapid, progressive changes which the *T*-complex undergoes. Similar changes may occur in pericarditis but this can usually be ruled out on clinical grounds.

From the electrocardiographic standpoint there are many types of infarcts which differ one from another as regards

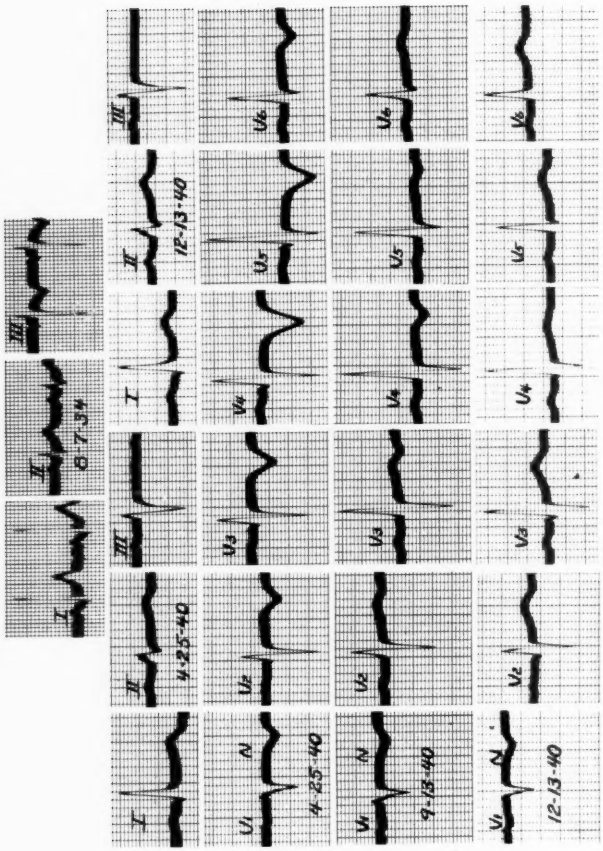


Fig. 22

Anterior myocardial infarction; changes in the *T* waves only.

the character of the electrocardiographic changes and the leads in which they occur. Whether some of these varieties are determined by the position of the heart rather than the location and extent of the infarcted region only the future can decide.

T-wave changes only. The electrocardiograms reproduced in Fig. 22 are those of a physician, aged 67 years, who was first seen on August 7, 1934. At that time the standard limb leads showed a considerable shift of the electrical axis to the left, but no other notable features. Myocardial infarction occurred early in March, 1940. Six weeks later (April 25, 1940) there was sharp inversion of the *T* deflections in all of the precordial curves, although the limb leads showed no significant changes. After eight months all electrocardiographic evidence of infarction had completely disappeared. The patient made an uneventful recovery. We have seen no instance of serious cardiac impairment following anterior infarction which produced no electrocardiographic abnormalities other than sharp inversion of the *T* waves.

Transient QRS changes. The electrocardiograms reproduced in Fig. 23 are those of a man who began to have symptoms typical of coronary thrombosis at 6:40 p. m. on October 6, 1936, while he was under treatment for prostatic hypertrophy. On the following day the standard electrocardiogram showed extremely small *QRS* deflections and flat *T* waves in lead I. In the precordial leads there was a progressive diminution in the size of *R* as the exploring electrode was moved from the first to the fourth position, and this deflection was abnormally small in leads V_5 and V_6. A month later the *QRS* deflections were normal, but there was sharp inversion of the *T* waves in all of the limb leads and in the last four precordial leads. The patient's recovery was complete and uninterrupted.

It is apparent that in this instance the infarcted muscle was for a time incapable of responding to the excitatory impulse, but it was not dead, and subsequently recovered its excitability. Very pronounced alterations in the *QRS* com-

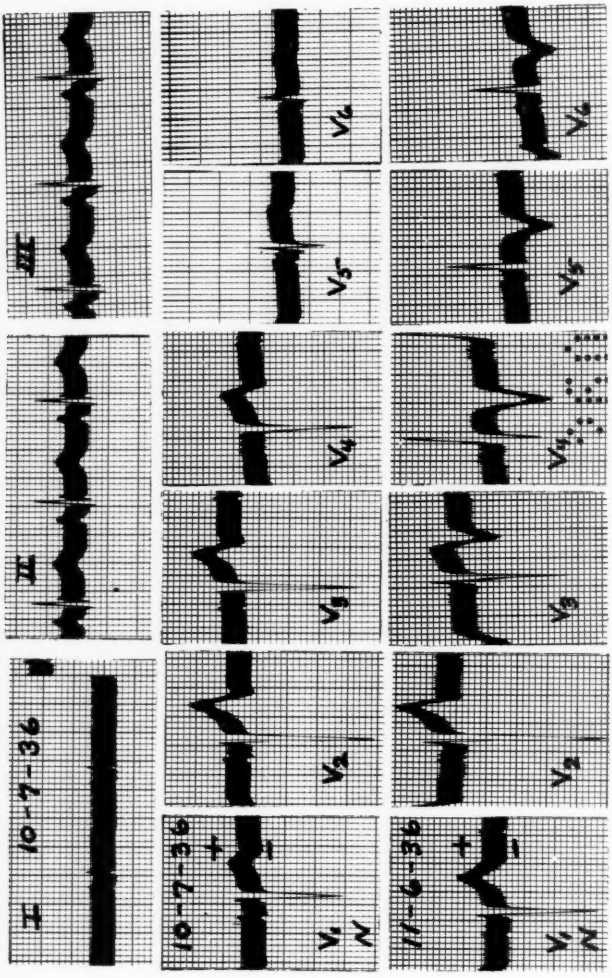


Fig. 23
Anterior myocardial infarction; transient *QRS* changes.

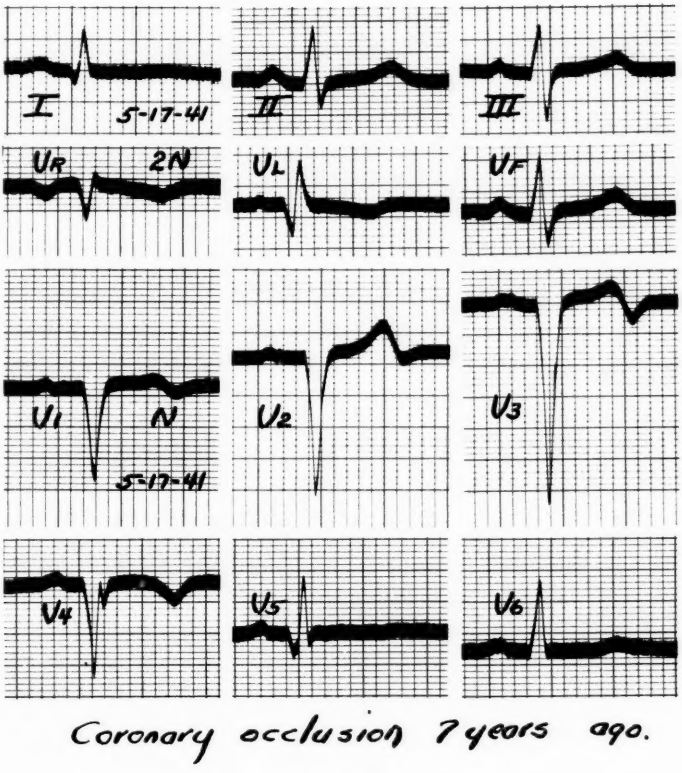


Fig. 24
Anterior myocardial infarction; persistent changes in the *QRS* complex and residual changes in the *T* waves.

plex of the kind observed in this case seldom disappear completely, but they often undergo a very slow and much less pronounced retrogression. Very often clear cut and characteristic deformities of this complex and much more rarely, residual changes in the *T* waves are still present

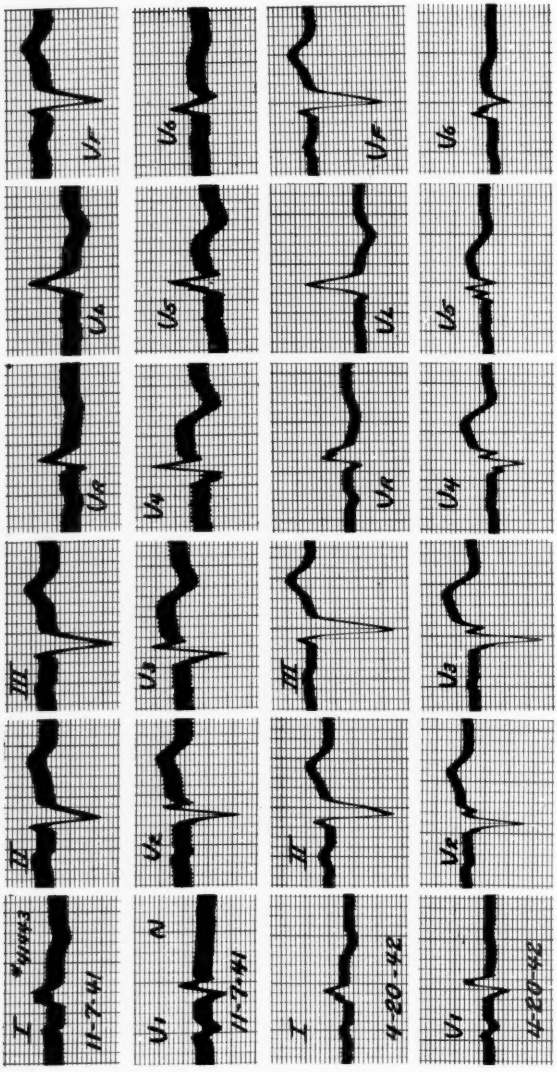


Fig. 25

Anterior myocardial infarction; persistent *RS–T* displacement, incomplete right bundle branch block.

years after the cardiac accident. An instance of this kind is illustrated in Fig. 24. The patient developed myocardial infarction in May, 1934, when he was 23 years old. The electrocardiograms taken at that time are not very different from those reproduced, which were made seven years later. When last seen this young man was working and was free of symptoms. Note that the ventricular complexes of the unipolar left arm lead are like those of lead V_5.

Persistent RS-T displacement. The electrocardiograms reproduced in Fig. 25 are those of a man, aged 73 years, who began to have anginal pain late in May, 1941, and developed myocardial infarction on June 11 of that year. Very pronounced *RS-T* displacement of the kind commonly seen in very recent anterior infarction was still present on November 7, 1941 when the first electrocardiogram was taken and remained essentially unchanged on April 20, 1942. The patient developed congestive cardiac failure but had chest pain only during the earliest stage of his illness. Complete healing of the infarcted muscle must have taken place long before the last curves were made. Ordinarily, pronounced *RS-T* displacement of the kind in question persists for a few hours, or at most for a few days. Why it persists in rare instances for weeks or months is still a mystery. In three cases of this sort, one of which was observed by Langendorf (20), a ventricular aneurysm was present, but it is possible that the association was due to chance. There is no known reason why ventricular aneurysm should displace the *RS-T* junction or deform the *RS-T* segment in this way. In addition to *RS-T* displacement both electrocardiograms display a *QRS* interval between 0.10 and 0.11 second in length and late *R* waves in the leads from the right side of the precordium. These findings strongly suggest that incomplete right branch block was present.

Extensive anterior infarction. In the three cases illustrated in Fig. 26 characteristic signs of infarction were present in all of the six precordial leads, or in all except the first. In

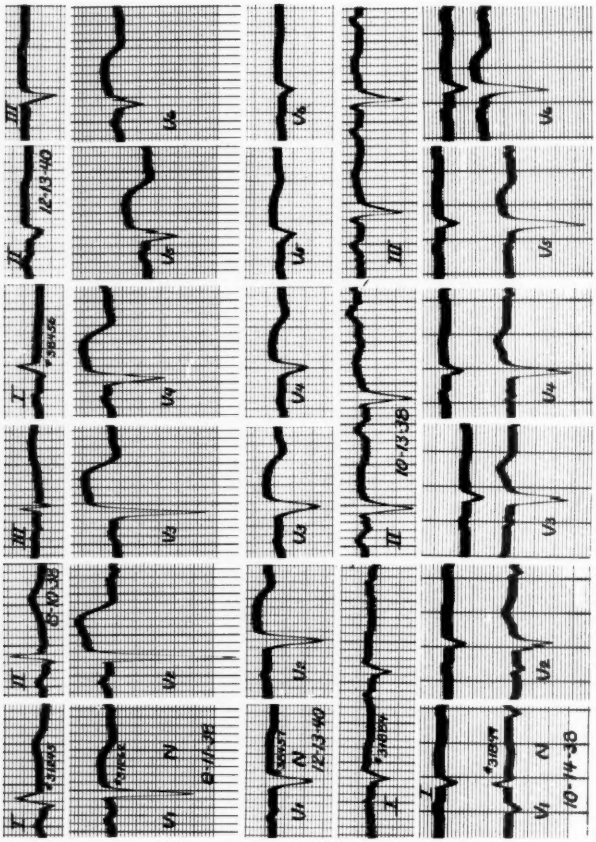


Fig. 26
Three cases of extensive myocardial infarction.

such instances it must be assumed that the region involved is a very large one. The first patient was a woman, aged 48 years, who began to have symptoms typical of coronary thrombosis at 9:45 p. m. on August 9, 1938. On the next day small *QRS* deflections, prominent *Q* waves, and upward displacement of the *RS-T* junction were present in lead *I*. On August 11 the precordial electrocardiogram displayed large *QS* deflections and conspicuous upward *RS-T* displacement in all leads except V_1. The second patient was a man, aged 71 years, who began to have severe pain in the chest at 9:45 p. m. on December 10, 1940. On December 13, the changes in the precordial leads were almost identical with those that occurred in the case just described. Both patients recovered.

The third patient was a man, aged 48 years, who had severe attacks of anginal pain on February 1 and on May 30, 1938. The first electrocardiogram was taken on October 14, 1938. At that time there were very large *Q* deflections in lead I and large *QS* deflections in the last five precordial leads. Probably because the infarct was an old one, no pronounced *RS-T* displacement or typical *T* wave changes were present. The patient died on October 16, 1938 and autopsy disclosed an infarct measuring 9 by 10 cm. which involved the lower two-thirds of the anterolateral and the lower one-third of the posterior wall of the left ventricle. The cavity of this chamber was almost completely filled by a thrombus.

Anteroseptal infarction. In the four cases illustrated in Fig. 27 the diagnostic signs of infarction (characteristic *QRS* combined with characteristic *RS-T* or *T* wave abnormalities) were confined to the leads from the right side of the precordium. Leads V_5 and V_6 display alterations in the *T* waves which are more or less typical but no unequivocal changes in the *QRS* complexes. Since the potential variations of the left arm are like those of the left side of the precordium in the majority of the cases of anterior myocardial infarction the changes in the ventricular complexes of lead I are as a rule no greater than those displayed by the ventricular complexes of lead V_5. An

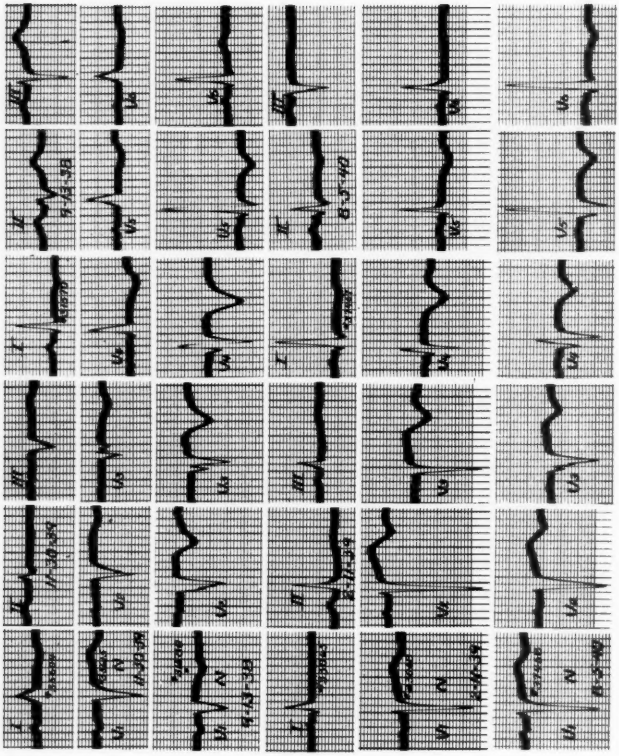


Fig. 27

Four cases of anteroseptal infarction.

electrocardiographic diagnosis of anterior infarction cannot, therefore, be made with certainty in cases of the kind under consideration unless leads from the right side of the precordium are employed. Since the potential variations of the infarcted region are referred to the right side of the precordium we may assume that the anteroseptal wall of the left ventricle is involved.

The first patient was a man, aged 68 years, who developed myocardial infarction in September, 1939. The electrocardiogram taken on November 30 of that year shows notched *QS* deflections in leads V_2 and V_3 and inversion of the *T* waves, not of the kind characteristic of infarction, in leads *I*, V_3, V_4, V_5 and V_6. The ventricular complexes of lead *I* closely resemble those of lead V_6.

The second patient was a man, aged 57 years, who had a coronary accident about September 1, 1938. The electrocardiograms made on September 13 show characteristic changes in the *T* waves of leads *I*, V_2, V_3, V_4 and V_5. Characteristic changes in the *QRS* complexes are confined to leads V_2 and V_3. The ventricular complexes of lead *I* are like those of lead V_5.

The third patient was a woman, aged 48 years, who began to have symptoms typical of coronary thrombosis on February 9, 1939. The electrocardiograms taken on February 11 show signs diagnostic of infarction in lead V_3 and typical *T* wave changes in lead V_4. The ventricular complexes of lead *I* are like those of lead V_6.

The fourth patient was a man, aged 59 years, who had a coronary occlusion on May 14, 1940. The electrocardiograms of August 5, 1940 display signs diagnostic of infarction in leads V_3 and V_4 and characteristic *T* wave changes in leads V_5 and V_6. The ventricular complexes of lead *I* are like those of lead V_6.

In normal subjects the *R* deflection steadily increases in height as the precordial electrode is moved from the first to the fourth

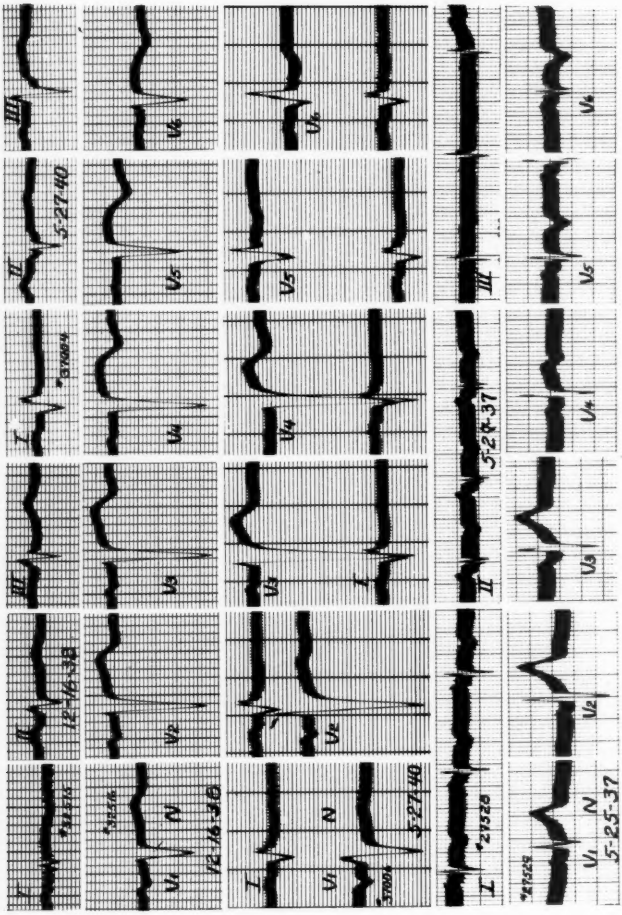


Fig. 28
Two cases of anterolateral and one case of posterolateral infarction.

position and then decreases as it is moved farther to the left. The decrease in the height of R and its eventual disappearance as the exploring electrode was moved across the right side of the precordium in a number of the cases just described is a far more reliable sign of infarction than complete absence of this deflection in the first two or first three precordial leads.

Anterolateral infarction. In the first two of the three cases illustrated in Fig. 28 the diagnostic signs of infarction in the chest leads are confined to the leads from the left side of the precordium, and it may be assumed that the anterolateral wall of the left ventricle was involved.

The first patient was a man, aged 70 years, who had a severe attack of anginal pain on July 6, 1938. The precordial electrocardiogram taken on December 16 of the same year displays signs diagnostic of infarction in leads V_4 and V_5, and abnormally small R deflections associated with changes in the T complex in lead V_6. In lead I the QRS deflections are extremely small and suggestive changes in the T waves are present.

The second patient was a man, aged 47 years, who was disoriented when admitted to the hospital, so that no history of his illness could be obtained. It was learned later that he had had anginal pain, but the time at which coronary thrombosis occurred could not be determined. The electrocardiogram displays deep Q deflections in lead I and in lead V_5 and the ventricular complexes of these leads are very much alike. There is also a rapid decrease in the size of the R deflection between lead V_2 and V_5. No characteristic modifications of the RS-T segment or T wave are present, probably because the infarct was an old one.

In infarcts of this kind there are, as a rule, changes diagnostic of infarction in the ventricular complexes of lead I. Since the infarct involves the left upper margin of the heart the potential variations at its epicardial surface are transmitted to the left arm.

The third case illustrated in Fig. 28 belongs to a different group and will be referred to later.

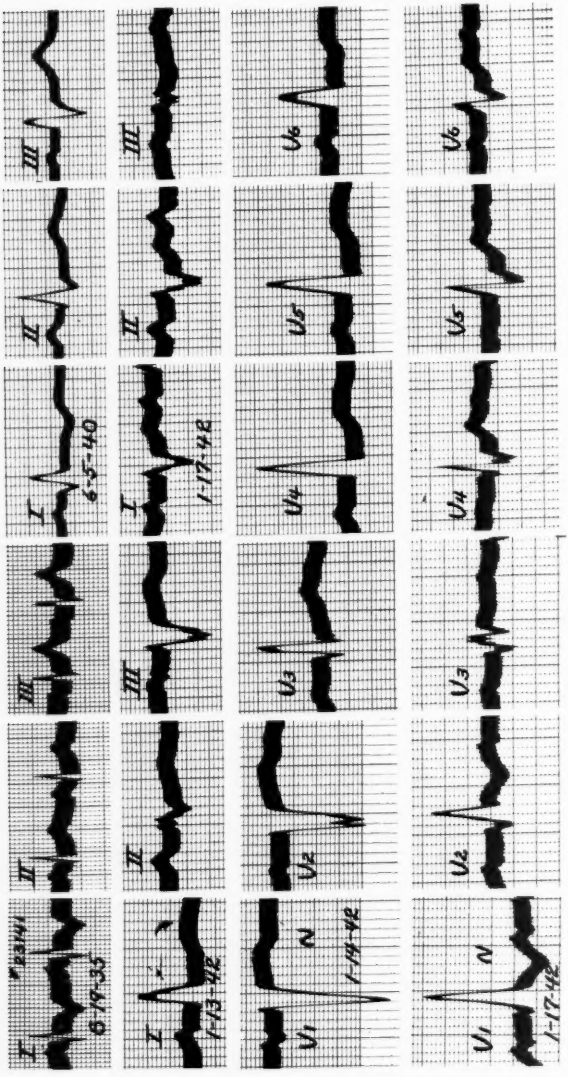


Fig. 29
High lateral infarction (see text).

High lateral infarction. The electrocardiograms reproduced in Fig. 29 are those of a man who was under observation off and on from September 1926 until his death on January 19, 1942. In the autumn of 1934 he began to have mild anginal pain on exertion after meals and at 3 a. m. on the morning of August 18, 1935, he developed symptoms characteristic of coronary thrombosis. The electrocardiogram taken on the following day presents small *QRS* deflections, deep *Q* waves, and sharply inverted *T* waves in lead *I*. The precordial electrocardiogram taken at the same time, which is not reproduced, is not definitely outside normal limits, but the *R* and *T* waves of leads V_1 and V_2 are unusually prominent. The patient had no more anginal pain and got along very well until June 3, 1940 at 12:30 p. m. when a second coronary accident occurred. The electrocardiogram taken two days later is very similar to that of 1935 except that the QRS interval is a little longer. Precordial leads were not used on this occasion. In October, 1941, paroxysmal nocturnal dyspnoea began to occur, and in January, 1942, the patient entered the hospital with the symptoms and signs of congestive cardiac failure, and attacks of substernal oppression.

On January 13, the limb leads showed a *QRS* interval measuring approximately 0.11 second, left axis deviation, and inverted *T* deflections in lead *I*. The precordial curves of the next day present a very small *R* deflection in lead V_2 and prominent *Q* waves in lead V_3, but no other changes which suggest infarction. The third attack of severe anginal pain began at 7 a. m. on January 15. On January 17, right bundle branch block was present. The precordial leads then showed changes characteristic of this conduction defect and small bizarre *QRS* deflections in lead V_3.

The autopsy disclosed a very large thrombus covering the apical two-thirds of the inner aspect of the anterior wall of the left ventricle. Beneath this clot the endocardium was grayish-white in color and near the base of the ventricle it was about one millimeter thick. Mottled grayish-pink to bluish-red areas were scattered through the anterior wall

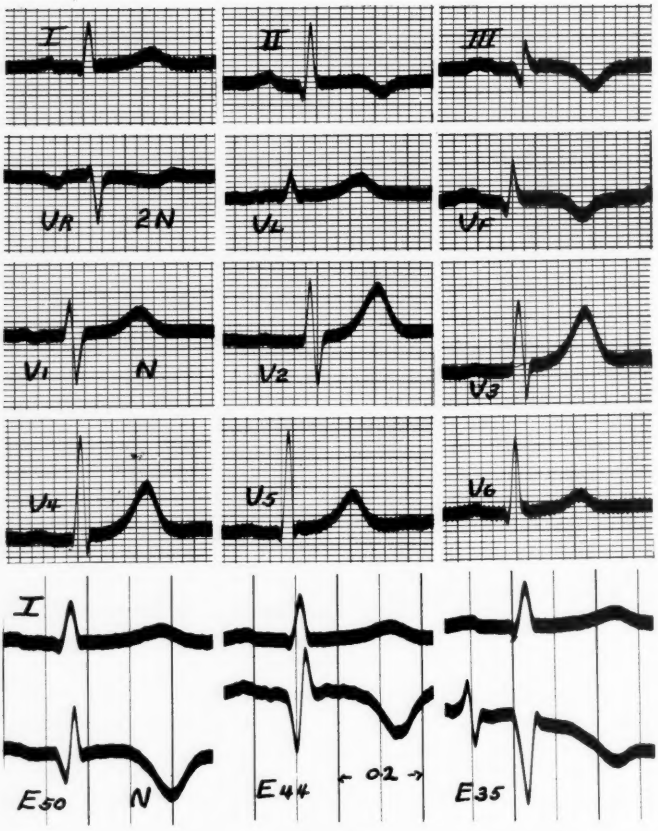


Fig. 30

Plain posterior infarction. In this as in other figures the symbol *E* is used for unipolar esophageal leads. The figure which follows this symbol gives the distance (in centimeters) of the esophageal electrode from the nostrils.

of the left ventricle, the septum, and the apical parts of the right ventricle, which also contained a thrombus. The left coronary artery was blocked by a fresh clot near its origin. In the postero-lateral wall of the left ventricle near its base there were extensive grayish-white to grayish-yellow areas which were thought to represent old myocardial infarction. The infarct responsible for the electrocardiographic changes observed in 1935 and 1940 apparently involved this region. We have seen only one other instance in which the limb leads showed signs diagnostic of anterior infarction and the precordial leads did not.

Plain posterior infarction. The man, aged 45 years, whose electrocardiograms are shown in Fig. 30 was referred to us for laboratory studies only. He had symptoms typical of coronary thrombosis, about one month before these curves were taken. The deep Q waves and sharply inverted T waves of leads *II* and *III* indicate that there was a recent infarct on the posterior wall of the heart. Note that the potential variations of the outer surface of the infarct were transmitted to the left leg. The precordial leads show no significant abnormalities, but it will be observed that in leads V_1 and V_2 the R deflections and T waves are unusually tall. Characteristic signs of infarction are present in the leads from the ventricular levels of the esophagus (leads E_{44} and E_{50}). In the lead from the auricular levels of the esophagus (lead E_{35}), there is a large diphasic auricular complex and the ventricular complex consists of a large QS deflection followed by an inverted T wave. Electrocardiograms of this kind are obtained from the auricular levels of the esophagus in normal subjects (21). The exploring electrode is close to the auricular wall which accounts for the character of the auricular complex and also opposite the great valvular orifices at the base of the ventricles so that the negativity of the ventricular cavities is transmitted to it. Large Q or QS deflections in leads from this region have no significance.

In the earliest stages of infarction of the posterior wall of the heart downward displacement of the RS-T junction is usually

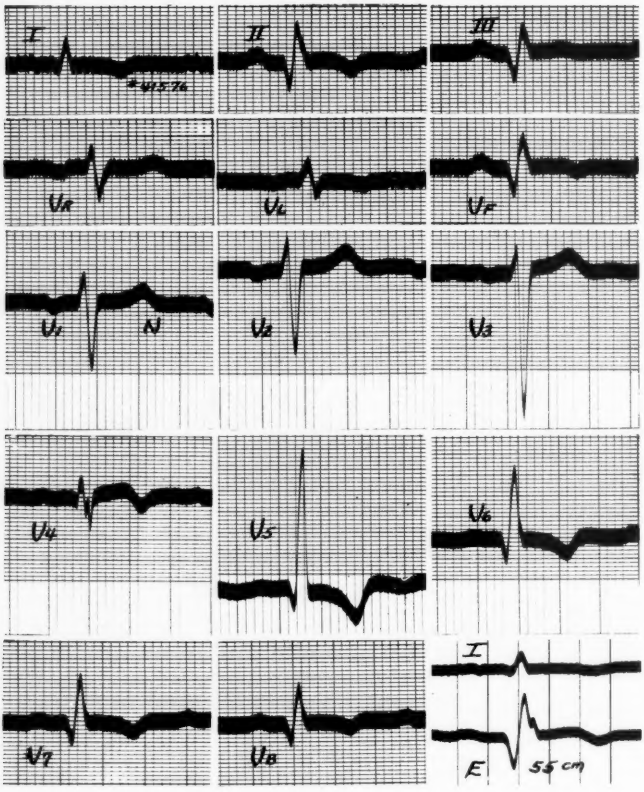


Fig. 31

Posterolateral infarction.

present in the precordial leads. In leads from the ventricular levels of the esophagus the displacement is upward (21).

Posterolateral infarction. The last of the three patients whose electrocardiograms are reproduced in Fig. 28 was a physician, aged 42 years, who had a coronary accident on March 18,

1937. The limb leads taken on May 25 of the same year display inverted *T* waves in leads *I* and *II* and small *QRS* deflections with prominent *Q* waves in leads *II* and *III*. There is also a small *Q* deflection in lead *I*. The changes in the *T* waves resemble those found in anterior, and the changes in the *QRS* complex those found in posterior infarction. In the precordial curves the significant abnormalities are in leads V_5 and V_6 which show prominent *Q* and sharply inverted *T* waves. It will be noted, however, that in the leads from the right side of the precordium the *R* and *T* deflections are unusually prominent.

Another electrocardiogram of the same sort is reproduced in Fig. 31. The patient was a man, aged 54 years, who had symptoms typical of coronary thrombosis about one month before the electrocardiographic study was made. In this instance unipolar leads from the left posterior axillary line, from the left infrascapular region, and from the ventricular levels of the esophagus were taken and all of these show prominent *Q* and sharply inverted *T* waves. Similar but less characteristic changes are present in the leads from the left side of the precordium (leads V_5 and V_6). There can be little doubt that the infarct involved the posterolateral wall of the heart. Note that here also large *Q* and inverted *T* waves occur in the unipolar left leg lead. Not all cases in which there are sharply inverted *T* waves in lead *I* and abnormally large *Q* waves in leads *II* and *III* are examples of posterolateral infarction. This electrocardiographic pattern in the limb leads may be produced by the simultaneous presence of two infarcts, an old lesion on the posterior and a more recent one on the anterior wall of the heart. It may also occur in anteroseptal infarction in which, perhaps because of some peculiarity in the position of the heart or because there is more involvement of the septal wall of the left ventricle than is usual, the potential variations of the central part of the infarcted ventricular wall are sometimes transmitted to the left leg.

Posteroinferior infarction. The electrocardiograms reproduced in Fig. 32 are those of a man, aged 53 years, who had symptoms

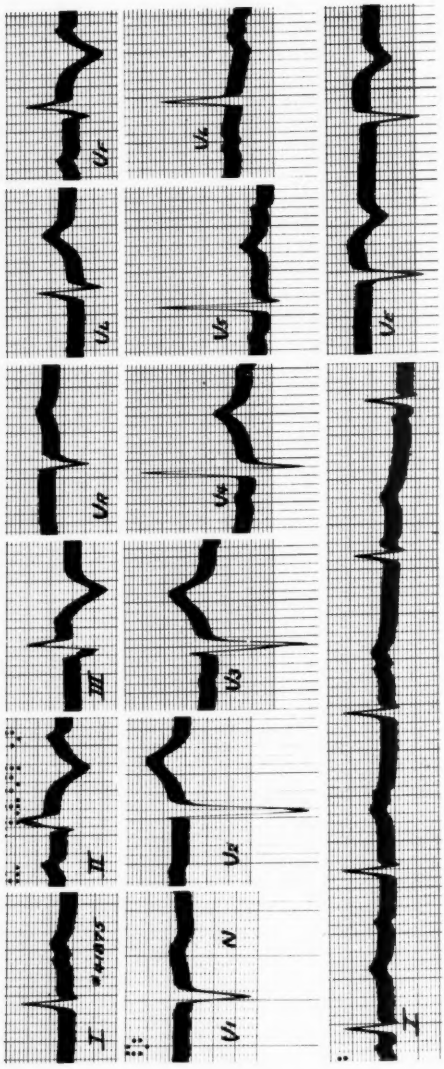


Fig. 32
Posteroinferior infarction.

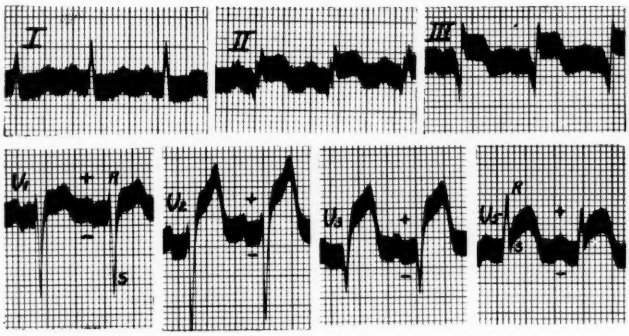


Fig. 33

Signs of posterior infarction in the limb leads and of anterior infarction in the precordial leads. (After Wilson (22).) Reproduced with the permission of the Macmillan Company, New York.

typical of coronary occlusion on December 19 and December 23, 1941. The curves taken on December 26 show upward displacement of the *RS-T* junction, sharply inverted *T* waves and prominent *Q* deflections in leads *II*, *III* and V_F, and complete atrioventricular block, which was transient. Signs diagnostic of infarction are present in the unipolar lead from the tip of the ensiform cartilage (lead V_E), but in none of the usual precordial leads. It will be noted that in this instance the *R* and *T* waves are not unusually prominent in the leads from the right side of the precordium. On the contrary, no *R* wave is present in lead V_1 and the *R* wave in lead V_2 is unusually small. In some cases of this sort slight upward displacement of the *RS-T* junction occurs in lead V_1 in the early stages of infarction.

Signs of posterior infarction in limb leads and of anterior infarction in the precordial leads. We have seen only one case of the kind illustrated in Fig. 33. The patient, a man aged 68 years, had a coronary accident on March 31, 1935, while he was under treatment for prostatic hypertrophy. He died three days later, but permission for an autopsy could not be

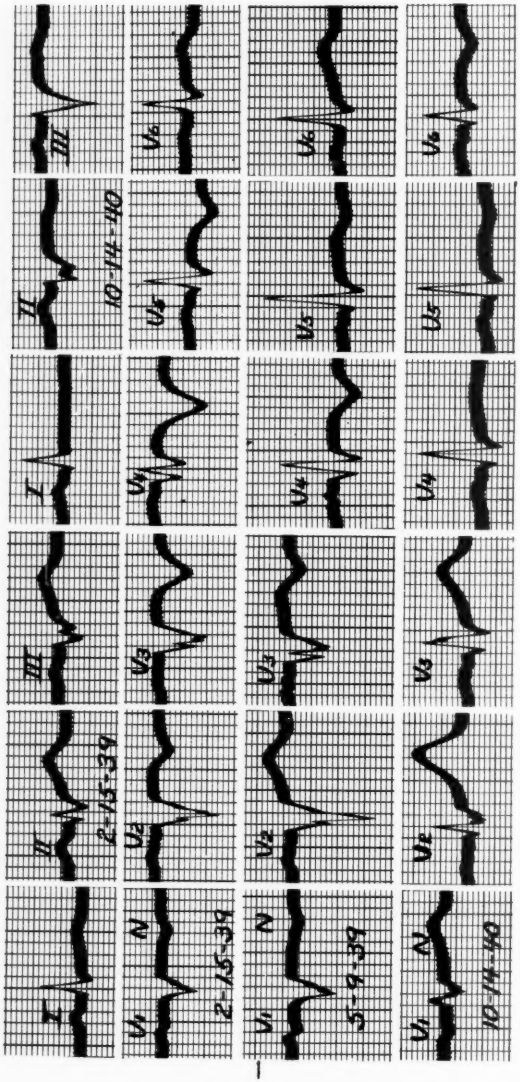


Fig. 34
Anteroseptal followed by posterolateral infarction.

obtained. No symptoms referable to the heart had occurred previous to this fatal illness. The limb leads show pronounced upward displacement of the *RS-T* junction and prominent *Q* deflections in leads *II* and *III* and in precordial lead V_3. There is, in adddition, conspicuous *RS-T* displacement without characteristic alterations of the *QRS* complex in leads V_2 and V_5. Leads V_4 and V_6 were not taken.

The character of the electrocardiographic changes precludes the possibility that there were two infarcts differing in age. Nor does it appear consistent with the presence of a single infarct extending from the posterior wall of the heart around the left margin onto the anterior wall, for the most striking changes in the precordial leads are in the middle of the series. We are not able to explain these findings satisfactorily, but may point out that apical infarction might give rise to electrocardiographic changes of this sort if the heart were in the vertical position.

Old anteroseptal and recent posterolateral infarction. The electrocardiograms reproduced in Fig. 34 are those of a man, aged 52 years, who had symptoms characteristic of infarction on December 18, 1938, and made an uneventful recovery. On August 29, 1940, he had a second attack of severe anginal pain. On this occasion he was examined at his home and the cardioscope disclosed very pronounced upward *RS-T* displacement in leads *II* and *III*.

The electrocardiograms taken two months after the first attack show prominent *Q* deflections but no characteristic *T* wave changes in leads *II* and *III*. The precordial curves of the same date display signs diagnostic of infarction in leads V_2, V_3 and V_4, and sharply inverted *T* waves in lead V_5. Note that the ventricular complexes of lead *I* resemble those of lead V_6, which are in no way abnormal. The infarct was, then, of the anteroseptal variety. I have already mentioned that infarcts of this kind may give rise to prominent *Q* deflections in leads *II* and *III*. After the second infarction sharply inverted *T* waves appeared in the complexes of lead V_6 which had previously escaped modification and initial *R* deflections were present in leads *II*

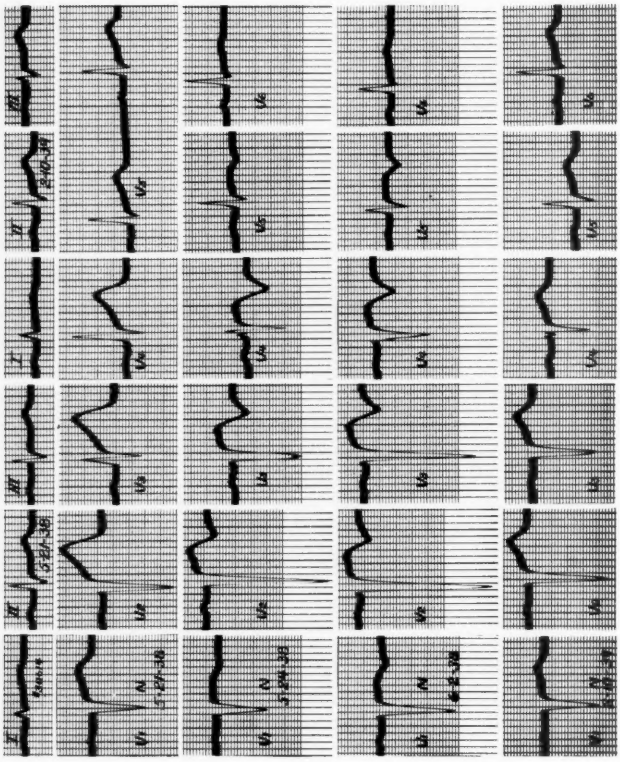


Fig. 35

Serial electrocardiograms in a case of anteroseptal infarction.

and *III*. At the same time conspicuous *R* waves were found in the leads from the right side of the precordium in which only embryonic *R* deflections had previously been present. Note, however, that the resurgent *R* waves are preceded by *Q* deflections in leads V_2, V_3 and V_4. Observe also the large size of the *T* waves in leads V_1, V_2 and V_3, in which deeply inverted *T* waves were present after the first coronary accident. The second infarct was, then, of the posterolateral kind.

We were able to make a more detailed study of another case in which the same series of events occurred. The patient was a man, aged 48 years, who had his first coronary thrombosis on May 21, 1938. The first electrocardiogram was taken about two hours after the onset of characteristic symptoms (Fig. 35). The limb leads showed no significant changes, but there was very pronounced upward displacement of the *RS-T* junction in leads V_2, V_3 and V_4. Three days later the *R* deflections had disappeared in leads V_1, V_2 and V_3 and had become very small in lead V_4, the *RS-T* displacement had begun to subside, and inversion of the terminal part of the *T* waves had developed in leads V_1, V_2, V_3, V_4 and V_5. The ventricular complexes of lead V_6 were not affected. In the course of the next year the *T* deflections regained their normal form, but the *QRS* changes persisted.

On March 5, 1942, the patient, who had been well for nearly four years, began to have new attacks of severe anginal pain. He was admitted to the hospital, but at first the electrocardiogram showed only the residual changes which dated from his first infarction (Fig. 36). On March 11, however, he developed clear signs of posterior infarction. In the extremity curves these consisted of pronounced upward displacement of the *RS-T* junction and prominent *Q* deflections in leads *II* and *III*. In the precordial electrocardiogram inverted *T* waves eventually developed in the complexes of lead V_6, which had escaped in the first attack, and small *R* waves appeared in leads V_1, V_2 and V_3. In these same leads the *T* waves became larger than they had been before.

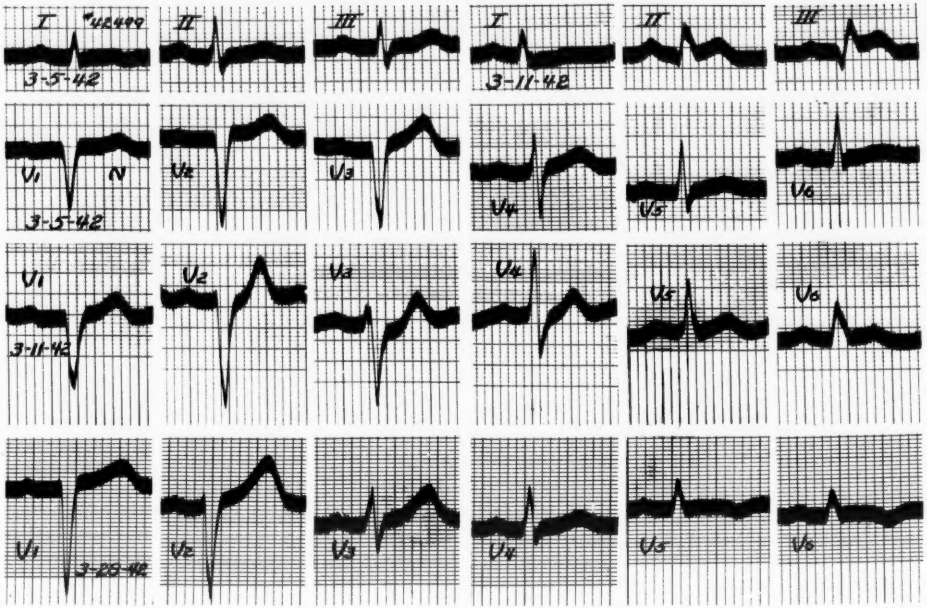


Fig. 36
Serial electrocardiograms showing posterolateral infarction following anteroseptal infarction. From the same case as Fig. 35.

Anterior infarction plus right bundle branch block. When anterior infarction produces right bundle branch block modifications of the electrocardiogram that can be regarded as diagnostic of infarction are relatively rare in the limb leads. Displacement of the *RS-T* junction and more or less characteristic changes in the *T* waves may be present but when the area of the *QRS* complex is large they are often obscured by the alterations in the *T* complex due to the block. In the precordial leads, on the other hand, entirely characteristic modifications of the *QRS* and *T* deflections are usually present. We have studied this problem experimentally by producing anterior infarction and right bundle branch block in dogs and there are no essential differences between this animal and man either as regards the character of the changes in the ventricular complexes of the limb leads or as regards the changes in the ventricular deflections of precordial leads.

The electrocardiograms reproduced in Fig. 37 are those of a man, aged 37 years, who had severe attacks of anginal pain on July 26 and 28, 1938. Two days after the last attack a pericardial friction rub was heard. The patient died on September 18, 1938, and autopsy disclosed thrombosis of the anterior descending coronary artery and a very large anterior infarct over which the pericardium had become adherent. Liquefaction necrosis of the central part of the infarct had taken place.

The limb leads taken on July 28 show small broad *QRS* complexes and slight upward displacement of the *RS-T* junction in lead *I*. The *QRS* interval measures approximately 0.12 second. The precordial curves taken on the following day are characteristic of anterior infarction complicated by right bundle branch block. The presence of the block is indicated by the length of the *QRS* interval, the very late *R* waves in leads V_1, V_2 and V_3 and the broad *S* waves in lead V_6. The small initial *R* deflections which occur in the leads from the right side of the precordium in uncomplicated right bundle branch block are absent. The presence of the infarct is indicated by the very large *Q* deflections in leads V_1, V_2, V_3 and V_4, the small bizarre *QRS*

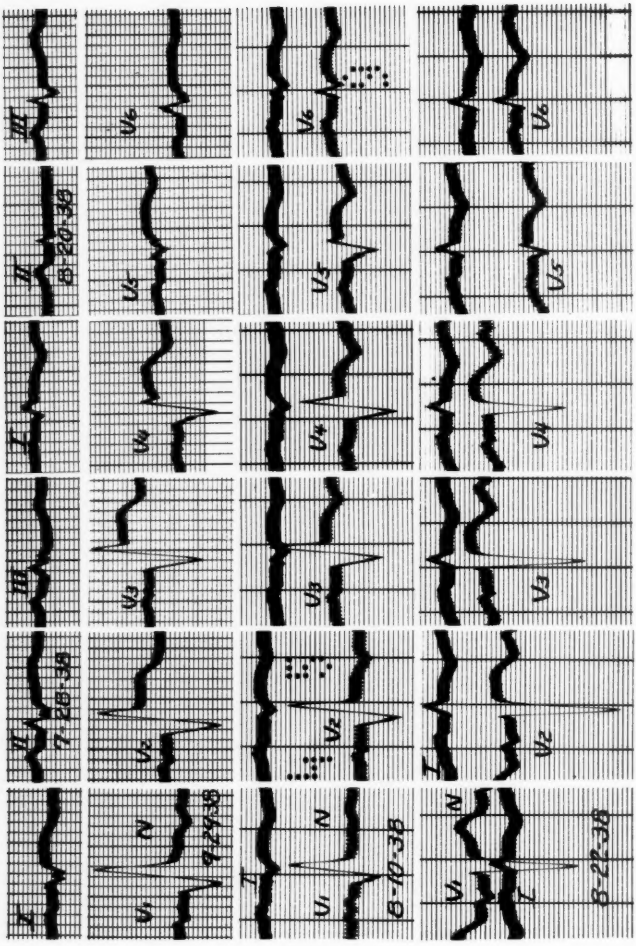


Fig. 37
Anterior infarction complicated by transient right bundle branch block.

deflections in lead V_5, and pronounced upward displacement of the *RS-T* junction in leads V_2, V_3 and V_4. The precordial curves taken on August 10 are not essentially different in general outline, but the RS-T displacement is not so great and the end of the *T* wave is slightly inverted in leads V_2 to V_6 inclusive.

The electrocardiograms taken on August 22 are very different. The *QRS* interval is normal and small *QRS* deflections, prominent *Q* waves, and sharply inverted *T* waves are present in lead *I*. In the precordial leads the *R* deflection decreases from lead V_1 to lead V_4 where it is absent and there are abnormally large *Q* deflections in lead V_5. Displacement of the *RS-T* junction and inversion of the end of *T* are present in leads V_2 to V_6. It is noteworthy that although initial *R* waves were not present in leads V_1, V_2 and V_3 so long as the branch block persisted they appeared when normal intraventricular conduction was re-established. It should also be noted that the *S* waves which follow these small initial *R* deflections are much deeper than the *Q* waves present in the same leads during block. These initial *R* waves must evidently be ascribed to forces produced by activation of the free wall of the right ventricle, and the deep *S* waves that follow them at least in part to the activation of septal muscle from the right side.

Another example of anterior infarction plus right bundle branch block is illustrated in Fig. 38. The patient was a man, aged 56 years, who had a coronary thrombosis either on September 23 or on October 3, 1938. The first electrocardiogram, taken before he had had anginal pain, is normal. After the coronary accident the limb leads disclosed the presence of right branch block but showed no changes characteristic of myocardial infarction. The precordial curves, on the other hand, are characteristic of both of these conditions.

Posterior infarction plus right bundle branch block. The electrocardiograms reproduced in Fig. 39 are those of a man, aged 53 years, who began to have symptoms characteristic of coronary thrombosis at 9 a. m. on April 30, 1941. The electrocardiograms taken on May 27, 1941 display large *Q* waves in leads *II*

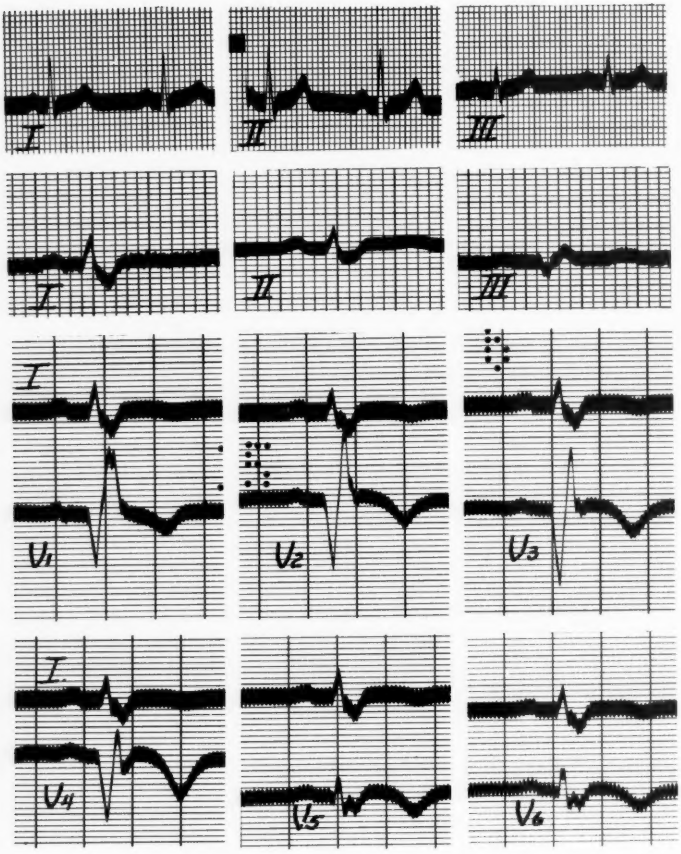


Fig. 38

Anterior infarction complicated by right bundle branch block.

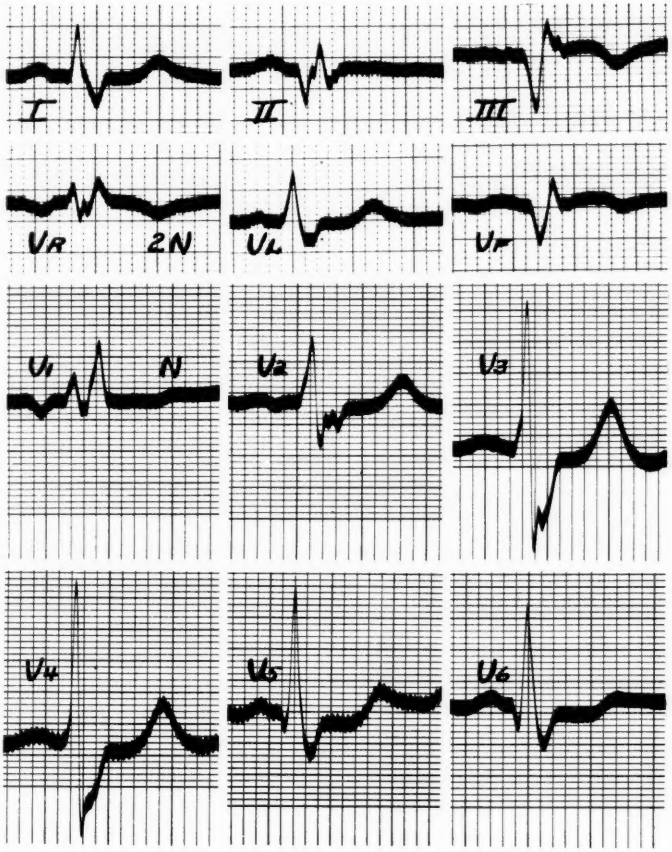


Fig. 39

Posterior infarction complicated by right bundle branch block.

and *III* and sharp inversion of the end of *T* in the latter. The *QRS* interval measures about 0.14 second and there is a broad *S* wave in lead *I*. The precordial electrocardiogram is characteristic of right bundle branch block. The conspicuous *Q* waves and *T* wave changes in lead V_6 suggest that the marginal parts of the infarct involved the lateral wall of the left ventricle. In the cases we have observed *QRS* changes in the limb leads characteristic of posterior infarction have not been obscured by the presence of right branch block.

Myocardial infarction complicated by "arborization" block. The electrocardiograms reproduced in Fig. 40 are those of a man who, after nine years of hypertension, developed myocardial infarction on August 28, 1941. In the electrocardiograms of May 5, 1942, the limb leads display a *QRS* interval of about 0.14 second, deep, broad *Q* waves followed by late *R* waves in leads *II* and *III*, and conspicuous *S* deflections in lead *I*. This electrocardiographic pattern is very similar to that shown in Fig. 39, and posterior infarction complicated by right branch block was suspected. The precordial electrocardiogram, however, is incompatible with the presence of this conduction defect, and shows no changes diagnostic of infarction. Nor is it characteristic of left branch block. The leads from the ventricular levels of the esophagus (E_{44}, E_{54} and E_{60}) present very broad, notched *Q* waves and very late, tall *R* deflections. It seems probable, therefore, that the infarct involved the subendocardial muscle on the posterior wall of the heart and not only permitted the negativity of the left ventricular cavity to be transmitted to its epicardial surface during the first part of the *QRS* interval, but also delayed the activation of less seriously injured outer layers of muscle which gave rise to the late *R* deflection.

The electrocardiograms reproduced in Fig. 41 are those of a man, aged 45 years, who had symptoms typical of coronary thrombosis in January 1938. The limb leads taken a year later, when the patient had congestive cardiac failure, display a *QRS* interval measuring between 0.11 and 0.12 second, small *QRS* deflections, and deep, broad *Q* waves in lead *I*. The curves from the right side of the precordium are like those seen in

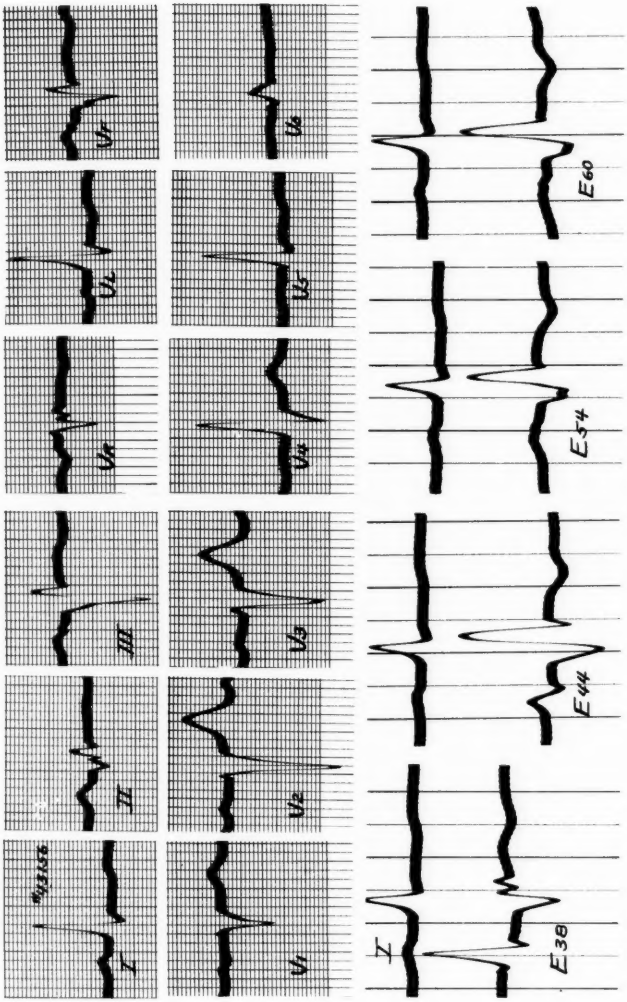


Fig. 40
Posterior infarction complicated by "arborization" block.

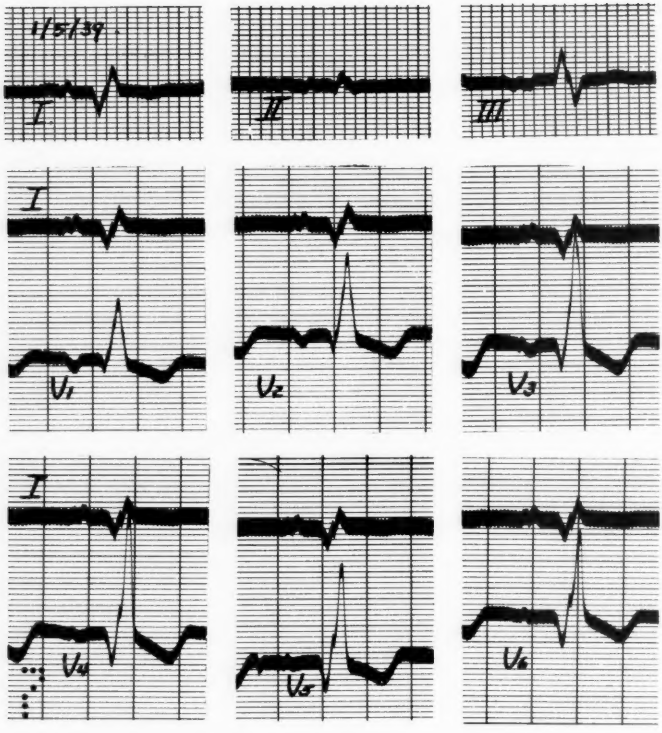


Fig. 41

Anterior infarction complicated by "arborization block", by right bundle branch block, or by both.

right bundle branch block complicated by anterior infarction, but in this instance the curves from the left side of the precordium are of the same type. It will be noted that these ventricular complexes closely resemble the ventricular complexes of the esophageal electrocardiograms reproduced in Fig. 40. In the case under consideration leads from the ven-

tricular levels of the esophagus yielded curves of the opposite type; the *QRS* complex of these tracings is diphasic and consists of an *R* wave followed by an *S* deflection of approximately equal size. It may be that these electrocardiograms represent right bundle branch block complicated by anterior infarction in which there is extreme displacement of the transitional zone. They are, however, more suggestive of anterior infarction with involvement of the subendocardial muscle over a large area complicated by delayed activation of more normal subepicardial muscle, and perhaps by right branch block as well. It seems probable that in some cases of old infarction the Purkinje network is damaged locally by subendocardial sclerosis and that as a result there is a delay in the activation of such living muscle as may remain in the part of the ventricular wall affected. Oppenheimer and Rothschild (*16*) had such lesions in mind when they introduced the term "arborization" block, but I doubt very much that the criteria for the electrocardiographic diagnosis of this condition advanced by them are capable of distinguishing between bundle branch block and interventricular block of other types.

In conclusion, I may say that we feel certain that in the long run unipolar precordial leads will be found superior to precordial leads of other kinds. They are the best available substitute for unipolar direct leads from the anterior surface of the ventricles. They are not directly influenced by the potential variations of the extremities; this makes them valuable in the analysis of electrocardiographic patterns in the standard limb leads with which we have long been familiar.

I must add that many views that I have expressed here must be regarded as tentative. There are still many problems to be solved. We do not know exactly what degree of correlation may exist between the electrocardiographic position and the anatomical position of the heart. We have, as yet, very little information as to the effect of the latter upon the precordial electrocardiogram. The factors that determine

the location and breadth of the transitional zone are still obscure. It is desirable that the effects of preponderant hypertrophy of the right and of the left ventricle upon the form of the precordial deflections be studied experimentally. It is still impossible to diagnose the lesser grades of this condition with any certainty. We need to have a better idea as to what parts of the heart's surface play the most important role in determining the potential variations of the extremities and other points on the body distant from the heart. We must study more carefully the electrocardiographic changes produced by combinations of lesions, such as right ventricular hypertrophy and right bundle branch block, and by conduction defects which delay the activation of local regions of muscle, particularly in myocardial infarction ("arborization block"). We must determine more exactly what the different electrocardiographic patterns seen in infarction mean in terms of the size of the infarct, its location, and the character of the changes in the muscle involved.

I feel sure that the future will see a great increase in the use of precordial and other special leads, and that it will bring us far greater knowledge of the electrocardiogram than we now possess.

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PRESIDENT WENSTRAND—Gentlemen, I have almost no words in which to express my feelings in regard to this very remarkable presentation. We have a little time, and it seems to me that there should be some questions or some comments from someone in the audience. Perhaps you all feel pretty much the way I do. Is there anyone who has anything on his mind?

DR. H. R. MAGEE—I would like to ask Dr. Wilson if the interesting changes in the *QRS* and *T* waves in precordial leads that he showed us in connection with the cases of cardiac hypertrophy, presumably some of those cases at least where the hypertrophy was due to hypertension or hypertensive heart disease—I would like to know if he feels that those electrocardiographic changes are probably caused by associated coronary sclerosis?

DR. REYNOLD C. VOSS—I would like to ask the Doctor if he would explain briefly just why it is that in the case of infarction, an infarction may cloud the picture of left bundle branch block, or, rather, why left bundle branch block may cloud the picture of an infarction?

PRESIDENT WENSTRAND—Any other questions? If not, Dr. Wilson, will you answer these questions?

DR. WILSON—In answer to Dr. Magee's question I may say that I do not believe that the changes in the *T* deflections often associated with left ventricular hypertrophy have been shown to be due to coronary sclerosis. The data available do not justify a final opinion as to their origin. I may point out, first of all, that in right ventricular hypertrophy large *R* waves and inverted *T* deflections often occur in the leads from the right side of the precordium. Although I have not compared these carefully with the large *R* waves and inverted *T* waves that occur in the leads from the left side of the precordium in left ventricular hypertrophy, it is my impression that there is no essential difference. In many of the cases of right ventricular

hypertrophy in which ventricular complexes of this kind occur there is no reason to suspect coronary disease. We must therefore consider the possibility that inversion of the *T* waves in ventricular hypertrophy is directly due to the increased thickness of the wall of the chamber chiefly affected. By delaying the activation of the subepicardial muscle the thickened wall also tends to delay its return from the active to the resting state, and therefore, to produce inverted *T* waves in unipolar leads from its surface and from adjacent parts of the body. When due to this cause the inverted *T* waves are directly dependent upon the increased area of the *QRS* complex and consequently upon the increased height and breadth of the *R* deflection with which they are associated, as is usually the case in bundle branch block. This is apparently not true in every instance of hypertensive heart disease in which inversion of the *T* waves in lead *I* occurs, for the inverted *T* waves sometimes become upright when the blood pressure is permanently reduced and the patient improves, even though the form of the *QRS* complex remains the same. It is possible that hypertrophy of the ventricular muscle leads to changes in its nutrition, even when the coronary arteries are not diseased, because of a disturbance in the relation of the blood supply to the quantity of muscle and the amount of work performed by it. One would expect inversion of the *T* waves, due to this cause, to be permanent unless we can assume that when the extra load is removed the hypertrophy undergoes retrogression, and if this occurred the *QRS* complexes as well as the *T* waves should become normal.

It has been observed in experiments on dogs that when the pulmonary artery is suddenly closed off, or greatly narrowed, by means of a clamp, *RS-T* displacement immediately develops in leads *II* and *III*. This must be attributed to acute injury and cannot be considered similar in origin to the persistent *RS-T* displacement often seen in left ventricular hypertrophy. It must be remembered that it is easier to modify the *T* complex than any other part of the electrocardiogram and that the factors that may alter the recovery process in the ventricular muscle are exceedingly numerous. I know that Dr. Arlie Barnes

of the Mayo Clinic uses the term "ventricular strain" in connection with the changes in the electrocardiogram that occur in hypertensive heart disease. I think that it is a great mistake to employ terms of this sort when discussing the electrocardiogram. The electrocardiographic deflections depend upon physiochemical processes, not upon mechanical phenomena. They can be interpreted only in terms of the spread and retreat of the cardiac impulse; no mechanical or anatomical expressions should be used in an electrocardiographic diagnosis unless an almost perfect correlation between the electrical phenomena and the mechanical or anatomical phenomena has been established beyond question, or the relations between the latter and disturbances of the spread or the retreat of the excitatory process are of a necessary and obvious kind.

In my opinion any departure from this principle will eventually bring discredit upon clinical electrocardiography as a science and will lead to endless confusion and disappointment. For this reason I believe, also, that the term "coronary disease" should never be used in discussing the interpretation of the electrocardiogram except in those instances in which the curves under consideration display changes diagnostic of myocardial infarction, or transient and unequivocal evidence of myocardial ischemia, if they have been taken during an attack of anginal pain.

In answer to the question asked by Dr. Voss I may say that when coronary thrombosis is complicated by left bundle branch block the electrocardiogram rarely presents changes which can be considered diagnostic of myocardial infarction. Displacement of the *RS-T* segment and changes in the *T* wave may occur if the area of the *QRS* complex is small. When the area of the *QRS* complex is large the alterations in the *T* complex due to infarction are likely to be obscured by those due to the conduction defect. The presence of characteristic modifications of the *QRS* deflections in infarction depends upon the transmission of the potential variations of the cavity of the left ventricle to the epicardial surface of the infarct and the ad-

jacent parts of the body. When this cavity is negative throughout the *QRS* interval, as is the case when intraventricular conduction is normal and in right bundle branch block, large *Q* or *QS* deflections occur in those leads in which one electrode is placed on that side of the left ventricle on which the infarct lies, provided that the connections are made in such a way that negativity of this electrode is represented by a downward deflection. In left bundle branch block the cavity of the left ventricle is positive at the beginning of the *QRS* interval and consequently *Q* or *QS* waves do not occur in leads of the kind mentioned. Direct or semidirect leads from the infarcted wall or from the cavity of this chamber display diphasic *QRS* complexes consisting of an *R* followed by an *S* deflection. Ventricular complexes of this sort are not sufficiently distinctive to have diagnostic value. When the septum is infarcted as well as the free wall of the left ventricle, the cavity of the left ventricle is initially negative, because the negativity of the cavity of the right is transmitted to it. Under these circumstances the electrocardiogram may display large *Q* or *QS* deflections in leads from the left precordium. The presence of large *Q* waves in these leads in left bundle branch block should always lead to the suspicion that the ventricular septum is involved.

A case of infarction which was complicated by left bundle branch block is illustrated in Fig. 42. The patient was a man, aged 56 years, who had a coronary occlusion on November 24, 1933. When he was first seen about ten days later the limb leads showed the changes usually observed in posterior infarction. Except for inversion of the *T* waves in lead V_6 the precordial leads were negative. A week later the patient developed transient intraventricular block with ventricular complexes similar to those observed in 1937 and 1938 (see Fig. 42). The area of the *QRS* group was small and the block did not abolish the inverted *T* waves in leads *II* and *III*. It did abolish the prominent *Q* waves in these leads. These curves have been published (Wilson (22) Fig. 78). When the patient returned in 1937 the intraventricular block was again present and it persisted. The ventricular complexes of the standard leads are of

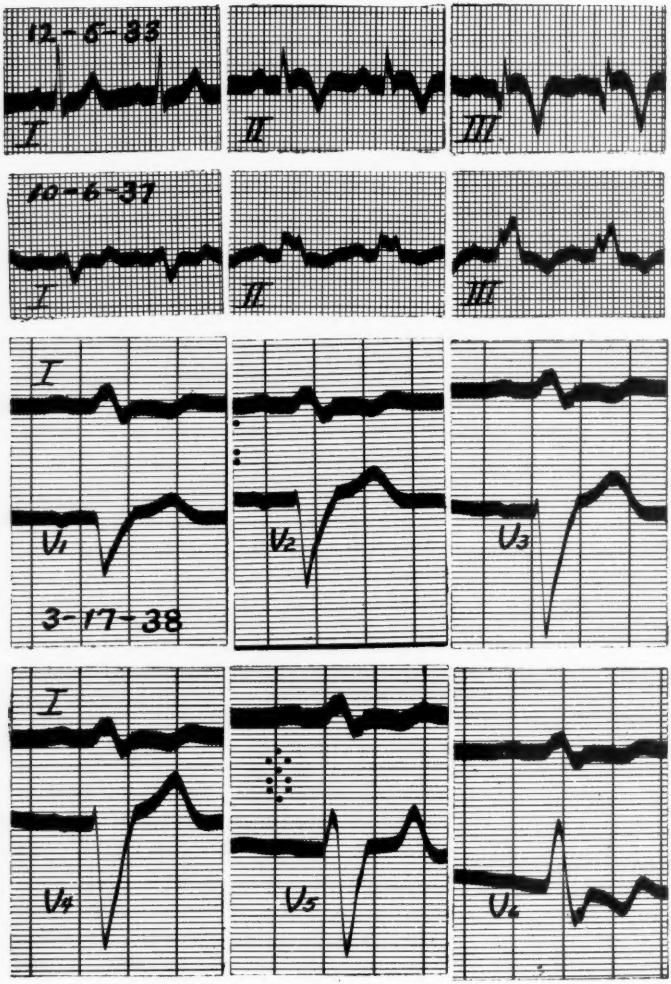

Fig. 42
Posterior infarction complicated by left bundle branch block.

the kind ascribed to right bundle branch block, but the precordial leads indicate that the conduction defect was in the left branch of the His-bundle instead of the right. The heart was in the vertical rather than in the horizontal position. The *QRS* complexes of lead V_6 are diphasic and, as regards their general outline, of the sort obtained by leading directly from the surface of an infarct of the left ventricular wall when left branch block is present. But *QRS* deflections of this kind may occur in lead V_6 merely because the transitional zone is shifted unusually far to the left. They are not sufficiently distinctive to be helpful in the diagnosis of infarction. A case of septal infarction with left bundle branch block is described in the article to which I have already referred (see Fig. 82 of that article).

PRESIDENT WENSTRAND—I hope we will have a return engagement at some time in the future.

It is just a little bit embarrassing for me to introduce the next speaker, who comes from my home office—not that I am ashamed of him or anything like that, but I shall only say that we have been interested in this subject for a good many years, and the contribution may be taken for what it is worth. Dr. Gamber F. Tegtmeyer!

THE SIGNIFICANCE OF SMALL NUMBERS OF MICROSCOPIC RED BLOOD CELLS and WHITE BLOOD CELLS IN THE URINE

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In recent years, clinical medicine has directed our attention to the significance of the microscopic cellular elements in the urine. Addis (1) in 1925 attempted a classification of Bright's disease on the actual number and combination of the formed elements in the urine. That same year, he pointed out that the presence of red blood cells, white blood cells and casts does not necessarily indicate disease (2). He states,

> "It is generally assumed that the presence of red blood cells in the urine is always indicative of some abnormality in the urinary tract, but this view is erroneous. On the other hand, they are probably not constant urinary constituents for I have sometimes failed to find them in spite of prolonged search in normal individuals in whose urine they have previously been present."

Addis went so far as to determine what he considered the normal range of excretion of red blood cells and white blood cells as well as casts in a period of twelve hours; namely, from none to 425,000 per twelve-hour period for red blood cells and from 32,400 to 1,835,000 per twelve-hour period for white blood cells.

The importance of albumin and casts to medical selection was stressed early. Davis (3) read a paper at the second annual meeting of the Association of Life Insurance Medical Directors on *Albuminuria in Persons Apparently Healthy and a Consideration of Its Relation to Life Insurance.* It was only following Ogden's (4) paper on *Urinary Impairments* in 1915 that the committee on *Nomenclature of Urinary Impairments* (5) in 1916 set up the table for the recording of red blood cells and leukocytes:

Rare	5 — 10
Occasional	11 — 20
Few	21 — 30
Numerous	More than the above.

In 1930, Clark (6) in his paper on *Pyuria* pointed out how inaccurate is such a method of reporting findings and suggested the use of the Fuchs-Rosenthal counting chamber. How many Companies have adopted his suggestions, we do not know, but the reporting procedure still follows the method of coding by number of cells counted per high-power field.

Since 1904, the Medical Department of the Northwestern Mutual Life Insurance Company has kept a record of every abnormal urine specimen examined in the Home Office laboratory. To date, some 20,000 cards have been accumulated covering single and combinations of the various urinary impairments. We were interested in seeing if, in spite of our rather inaccurate method of recording—as pointed out by Clark—, we could find anything significant in our cards showing only red blood cells, white blood cells, or a combination of the two. Would our experience support Addis's postulate that individuals may show microscopic red blood cells or white blood cells normally? Was our underwriting sound in approving individuals who occasionally showed microscopic red blood cells and white blood cells? We examined, therefore, only approved lives from 1904 to 1940, carried to the anniversary in 1941, who at the time of approval showed in at least one specimen ex-

amined at the Home Office red blood cells, white blood cells, or a combination of the two. In all other respects, the cases were chosen with particular care. The lives studied totalled 1,676. The group included 138 women and 1,538 men. (Women have been issued insurance only since 1933.) There were no deaths among the women. Specimens were examined at the Home Office in about one-third of the group because of the amount of insurance applied for. In two-thirds of the cases, specimens were requested because of the past medical history or findings on examination in the field. The group therefore is weighted somewhat with amount cases, but not necessarily "Jumbo" risks as specimens are required in cases of $25,000 and over. The basis of the study was lives with the expected deaths calculated according to the American Men Select Table.

The cards were divided first on the basis of year of examination. See Table I for the result.

TABLE I

EXPERIENCE BY YEAR OF EXAMINATION

Year of Examination	No. of Lives	Actual Deaths	Expected Deaths	Ratio % Act./Exp.
1904 — 1920	301	88	94.9	92.7
1921 — 1940	1375	113	115.4	97.9
Total	1676	201	210.3	95.6

From 1904 to 1920, 301 lives with 88 actual deaths and expected deaths 94.9 give a mortality ratio of 92.7%. 1,375 lives appeared from 1921 to 1940 with 113 actual and 115.4 expected deaths giving a ratio of 97.9%. Laboratory examinations from 1904 to 1920 were made by physicians of the Home Office Staff —those from 1921 to 1940 by the technicians, but all positive findings were checked by a physician. The results are very similar with a slight but scarcely significant increase in mortality in the second group.

The cards were then divided according to age at the time of the positive findings. Table II is the result of the cards distributed according to age.

TABLE II

EXPERIENCE BY AGE AT EXAMINATION

Age at Examination	No. of Lives	Actual Deaths	Expected Deaths	Ratio % Act./Exp.
Under 40	798	38	47.1	80.7
40 & over	878	163	163.2	100.0
Total	1676	201	210.3	95.6

798 lives under age forty with 38 deaths as compared with an expected of 47.1 deaths gave a ratio of 80.7%. 878 lives over age forty gave 163 actual deaths as compared with 163.2 expected deaths, or a ratio of 100%. An occasional positive finding, therefore, appears more significant after age forty.

We attempted to study red blood cells in the urine on the basis of the M. I. B. division; namely, one to five, six to ten, and eleven or more cells. The second and third groups had so few deaths that we grouped all of the cards showing microscopic red blood cells into one division. In the 485 lives, there appeared 35 deaths against expected deaths of 34.8, giving a ratio of 100.6. (See Table III)

There were 1,109 lives in the white blood cell group with 157 deaths as compared with an expected of 163.8, or a ratio of 95.8%. These cards were separated on the basis of the M. I. B. division; namely, six to ten, eleven to twenty, and twenty-one and up cells. As the number of cells increased, there was a slight increase in the mortality, as one might expect. The group showing eleven to twenty cells gave 42 deaths with expected 40.7 deaths, or a ratio of 103.2%. Those showing twenty-one and up cells had twenty deaths against an expected 18.8 deaths, or a ratio of 106.4%. The combination of red blood cells and white blood cells appeared only 82

TABLE III

EXPERIENCE BY IMPAIRMENT

Impairment	No. of Lives	Actual Deaths	Expected Deaths	Ratio % Act./Exp.
Red Blood Cells	485	35	34.8	100.6
White Blood Cells:				
6–10 cells	598	95	104.3	91.1
11–20 "	391	42	40.7	103.2
21–up "	120	20	18.8	106.4
All	1109	157	163.8	95.8
R.B.C. & W.B.C.	82	9	11.7	76.9
Total	1676	201	210.3	95.6

Note: Estimated general experience of the Company under all policies of corresponding issue and exposure is 72% by policies.

times with nine deaths against an expected 11.7 deaths, or a ratio of 76.9%. In the total 1,676 lives, 201 deaths occurred where 210.3 were expected, giving a ratio of 95.6%. There is little significant variation in the ratio among the group save those showing the smallest number of pus cells are most favorable. The red blood cells and white blood cells combined because of the small number involved (82 cases and 9 deaths) cannot be considered of much significance.

While the mortality ratio may not appear significantly high, yet when it is remembered that these cases were selected with particular care so far as other impairments were concerned, the rather high figure appears more impressive. Secondly, when compared with the general experience of the Company under all policies of corresponding issue and exposure of 72%, the results are not satisfactory.

Ogden (7), in his discussion of Blakely's paper on *Urinalysis*, made this significant comment,

> "It is interesting to see how few cases with urinary impairments die of kidney diseases or important trouble in any part of the urinary tract."

ANALYSIS OF CAUSES OF DEATH IN 201 CASES SHOWING R. B. C.'S AND W. B. C.'S OR A COMBINATION FROM 1904 TO 1941, INCL.

TABLE IV

	20–29	30–39	40–49	50–59	60–69	70–79	80+	Total	%	1921 to 1941, incl.
Cancer	—	—	2	8	11	2	—	23	11.4	10.7
Tuberculosis	—	1	—	2	—	—	—	3	1.5	3.1
Diabetes	—	—	—	—	—	2	1	3	1.5	2.1
Syphilis	—	—	—	2	1	—	—	3	1.5	.2
Other General Diseases	—	—	—	6	6	1	—	13	6.5	5.8
Diseases of the Nervous System	—	—	—	—	2	1	—	3	1.5	3.8
Diseases of the Cardio-vascular System	—	2	7	20	32	20	2	83	41.3	38.3

TABLE IV Continued

	20–29	30–39	40–49	50–59	60–69	70–79	80+	Total	%	1921 to 1941, incl.
Respiratory Diseases	—	2	4	5	2	1	—	14	7.0	6.3
Digestive Diseases	—	—	2	2	6	2	—	12	6.0	7.4
Nephritis and Kidney Disease	—	1	—	3	5	3	—	12	6.0	5.4
Bladder and Prostate Diseases	—	—	—	1	—	2	—	3	1.5	2.5
Suicide	—	3	3	5	2	1	—	14	7.0	3.7
Casualties	1	2	3	4	4	1	—	15	7.5	7.5
Total Deaths	1	11	21	58	71	36	3	201	100%	
Ages at Death %	.5	5.5	10.4	28.9	35.3	17.9	1.5		100%	
% All Co. Deaths 1921 to 1941	4.9	10.01	15.66	21.9	24.26	16.85	6.07			

We attempted, therefore, to determine if in our 201 deaths there was any significant increase in diseases of the genito-urinary tract. We used as a control all of the deaths occurring in the Company from 1921 to 1941, as most of the deaths in our series occurred in these years. Table IV was obtained. Reference to it will disclose that there was apparently no significant trend in the causes of death. The suicide rate appears high, but nine of the fourteen deaths occurred during the depression years, when the general suicide rate was high.

Summary and Conclusion.

1,676 approved lives were studied showing red blood cells, white blood cells, or both in the urine at the time of the examination.

201 deaths occurred compared with a calculated 210.3 (A. M. Select) deaths, giving a ratio of 95.6%. The figure may appear significant when compared with the Company experience by policies of 72%.

In the selection of risks, the occasional finding of red blood cells or white blood cells appears to be more significant after the age of forty than before.

In spite of unusual care in the selection of the risk so far as other impairments are concerned, the presence of red blood cells or white blood cells (eleven or more) in an occasional specimen of urine gives an unfavorable mortality. Perhaps more attention to the actual number of cells present would be advisable. The more thorough urological study now available and employed in this type of case may yield more satisfying results.

No significant trends in the causes of death were found.

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PRESIDENT WENSTRAND—It is a pleasure to introduce to you Dr. Meredith Fairfax Campbell who was born at Lake Geneva, Wisconsin, which is a suburb of Milwaukee. It, therefore, seems appropriate that he should carry on more effectively from where our statistical paper ends. His medical education, training and practice centered in and around New York. I shall not mention schools and hospitals, except to say that Dr. Campbell is Professor of Urology at New York University Medical School, a Fellow of the American College of Surgeons and Lt. Commander, Medical Corps of United States Naval Reserve. His subject is, "Prognostic Considerations in Pyuria and Hematuria." Dr. Campbell!

DR. CAMPBELL—I was very much interested to hear my origin brought into this. I might say that I bought my first pair of long pants in Milwaukee.

PROGNOSTIC CONSIDERATIONS IN PYURIA AND HEMATURIA

By Meredith F. Campbell, M. D.

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In a splendid recent contribution on the "Significance of Small Numbers of Red and White Blood Cells in the Urine," Wenstrand and Tegtmeyer (1) indicated that this condition renders the prognosis only slightly unfavorable. These observations were made on 1676 (1538 male, 138 female) insured subjects whose urine at the time of their initial examination for insurance showed minimal pyuria or/and hematuria. On the actuarial basis of the American Men Select Table the calculated mortality was 210.3, whereas 201 (95.6 per cent) died. This 95.6 per cent is in contrast to the Company normal death expectancy of 72 per cent—a difference of 23.6 per cent. Moreover, in individuals showing pyuria and hematuria when first examined after forty years of age, the mortality was notably higher than in the group with normal urine (100 per cent of calculated as to 72 per cent). Yet only 7.5 per cent (about 1:14) of all deaths in the pyuria-hematuria group were attributable to urologic disease.

Upon studying these data, and particularly as they suggest the remarkably good life expectancy of adults with minimal pyuria or/and hematuria at the time of initial examination for insurance, my first reaction was that we urologists have perhaps over-estimated the unfavorable potential vital significance of these abnormal urinary findings. Yet I strongly suspect that in the reported group there were many gross errors due to examination of the specimens collected by methods open to criticism; this will be discussed elsewhere. In 1109, or 65.5 per cent of the total number of cases, white blood cells were the only abnormal urinary finding; in 485, or 28.8 per cent, the red blood cells constituted the urinary abnor-

mality; and in the balance of 82 cases both red and white blood cells were found. There is good reason to believe that by improvement in the method of urine collection the incidence of true pyuria would have been considerably lower, but the rate of actual to calculated mortality would have been proportionately higher. In short, pyuria and hematuria demonstrable in properly collected specimens has special vital significance. Grave urologic disease repeatedly is being demonstrated in patients whose urine contains these abnormal elements in no greater numbers than indicated in their paper (1). It is more than likely that the relative mortality of such of these patients as might be insured, is so small in mass consideration that the mortality is not seriously increased. It is a matter of the comparatively small clinical experience and horizon of an individual or even a clinic group with these particular cases as against the infinitely greater numbers of the insured group. Most of the patients we urologists and other clinicians examine because of gross or microscopic hematuria and/or pyuria, have clinical histories which at once render these individuals subjects for special scrutiny and, commonly, rejection by the propective insurance carrier. From the accumulated data of many large medical centers we know the approximate mortality incidence of the various urologic diseases in patients with hematuris and the even greater number with pyuria. This data is obtainable in textbooks on urinary tract disease and in special publications by various observers. Here we will concern ourselves only briefly with the commoner conditions which may produce hematuria and pyuria of all grades, together with their prognostic considerations, and will indicate certain common lesions which may produce minimal pyuria and/or hematuria, with special attention to acceptable methods of collecting the urine specimen.

Pyuria, hematuria and pain are the cardinal symptomatic triad of urinary tract inflammation which in turn usually results from infection. Pain and other subjective symptoms are not germaine to the present discussion; we are here interested

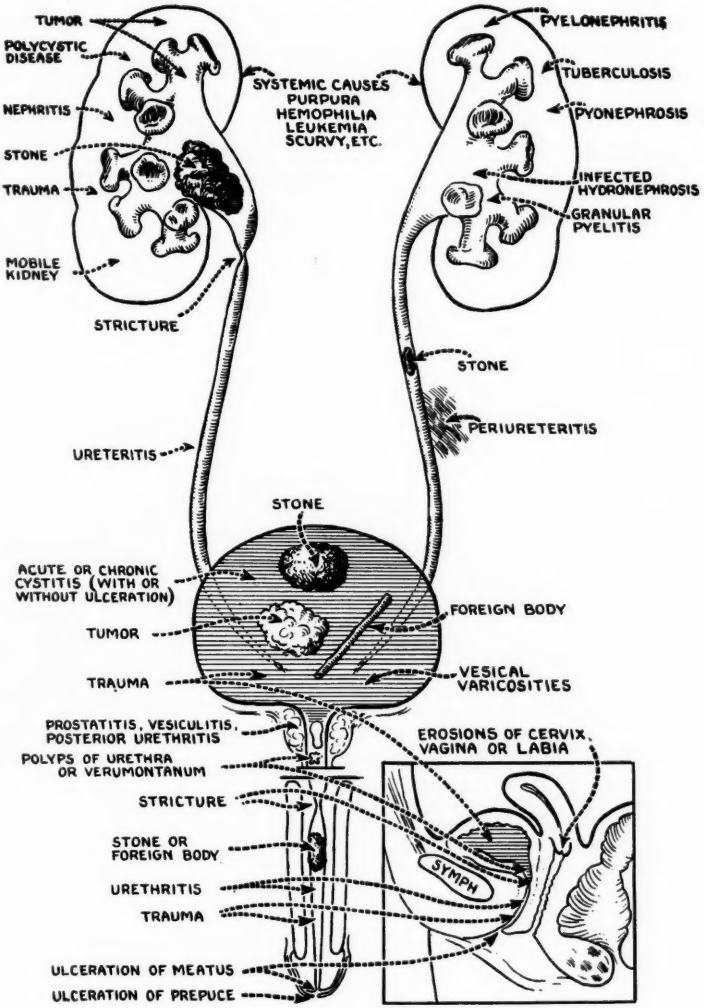

Fig. 2

Causes of hematuria. Only by urologic examination, surgical exploration, or autopsy can most of these lesions be identified.

only in the condition of the individual and his probable prognosis when the urinalysis report indicates the presence of pus and/or blood. As a collateral consideration, the presence of small amounts of pus or blood in the urine will give a variably positive albumin test and, in the interpretation of the albuminuria, the cytologic findings in the urine necessarily assume great importance.

PYURIA

The common causes of pus in the urine are schematically indicated in Fig. 1, where it will be noted that infection and obstruction commonly go together. Sterile pyuria is occasionally observed and for practical purposes should be considered tuberculous until proved otherwise. Yet some obstructions, stone, foreign body or instrumental reaction, may cause pyuria in the absence of infection.

Since pyuria and hematuria are so commonly due to the same lesion, the general prognostic considerations of these conditions will be discussed under hematuria. To repeat: The amount of pus in the urine is no criterion of the severity of the disease and an inexperienced microscopist may easily overlook an important but minimal pyuria. Most minimal pyuria is due to readily remedial causes, although in some instances it reflects a grave and incurable condition. Certainly the applicant who shows a minimal pyuria is entitled to an opportunity to have the condition corrected rather than to have his application rejected. For example, urinalysis of the honeymooning groom would often cause insurance rejection; congestive prostatitis with microscopic hematuria and pyuria is the explanation. Likewise the traumatic urethritis and urethrotrigonitis of his bride at this period would be likely to cause the appearance of a variable number of red and white blood cells in the urine. In many instances the eradication of infection or of a minor obstructive lesion, such as a tight external urethral meatus, is all the treatment that is required, can readily be accomplished and thus render

the applicant a sound risk from the standpoint of the insurance carrier.

Most pyuria results from urinary infection, generally arising in the urinary tract itself, and should always be considered a manifestation of renal infection until proved otherwise. Twenty-five per cent of patients with chronic pyelonephritis will die of renal failure and most of these within fifteen years. Moreover there is good reason to believe that much of so-called Bright's disease is in truth a bacterial nephritis with sclerosis, or is the sclerotic end result of an unrecognized antecedent bacterial nephritis. In reporting on a series of 2040 cases of chronic pyelonephritis observed at the Mayo Clinic and treated in the pre-sulfonamide era, yet subjected to the most favorable therapy of that time, Braasch and Cathcart (2) found that a third of the patients were cured, a third were improved and a third were unimproved. Certainly in the unimproved cases death from renal failure can be anticipated. In 1938 Braasch (3) reported a series of seventy-nine patients with chronic pyelonephritis treated by mandelic acid therapy with 27 (34 per cent) cured. On the brighter side, Alyea (4) in 112 cases of urinary infection treated by sulfathiazole found 71 per cent cured, 26 per cent improved and only 3 per cent were unimproved and he achieved essentially the same results in 72 cases treated by sulphapyridine. In short, sulphonamide therapy more than doubles the number of cures, with a striking decrease in the number of patients who show no improvement. This is readily translated into greatly improved prognosis.

When persistent pyuria is part of the picture of lower urinary tract obstruction which is unrelieved, all such patients may be expected to die of renal failure. Especially is this true of prostatic and urethral stricture obstructions, which are characteristically progressive. When the obstruction is in the upper urinary tract and unilateral and, if the function of the renal mate is preserved, the patient's outlook is decidedly improved. The prognosis depends upon whether the involve-

ment is unilateral or bilateral, as well as the character of the infection (e. g. tuberculosis) or other pathology such as renal tumor, obstruction (hydronephrosis) and so forth.

In renal tuberculosis 90 to 100 per cent of the patients show pus and blood in the urine. In Wildbolz's series of patients with unilateral tuberculosis it was found that without operation 42 per cent were alive after five years and 6 per cent were alive after 10 years. But in those patients with unilateral disease and subjected to nephrectomy, 55 per cent were alive after 10 years.

The conditions which are particularly likely to cause microscopic pyuria and/or hematuria in the absence of infection or striking urologic symptoms are ureteral and urethral stricture and urethrotrigonitis in the female, and prostatitis in the male. Yet ureteral stricture commonly causes pain in the side or reflex gastrointestinal disturbances leading to the diagnosis of intestinal indigestion, flatulence or biliousness, and so forth. Urethrotrigonitis usually causes frequency of urination. Yet the last condition is more commonly associated with urethral stricture, and both ureteral and urethral stricture respond to appropriate instrumental dilatation. In these conditions the urine may be sterile, crystal-clear and normal on analysis except for microscopic pus or blood.

Low-grade prostatitis causing microscopic pyuria and/or hematuria is extremely common. In most of these cases the etiology is non-venereal, predominantly staphylococcal, and is frequently metastatic from dental infection. Gastrointestinal foci are of next highest incidence. Patients with low-grade asymptomatic prostatitis are likely to show a variable quantity of pus in the urine and in a certain number of cases red blood cells; the last usually originates in the congested mucous membrane of the deep urethra covering the prostate and verumontanum. In short, in the presence of pyuria and/or hematuria the precise diagnosis can seldom be made without adequate urologic investigation which frequently re-

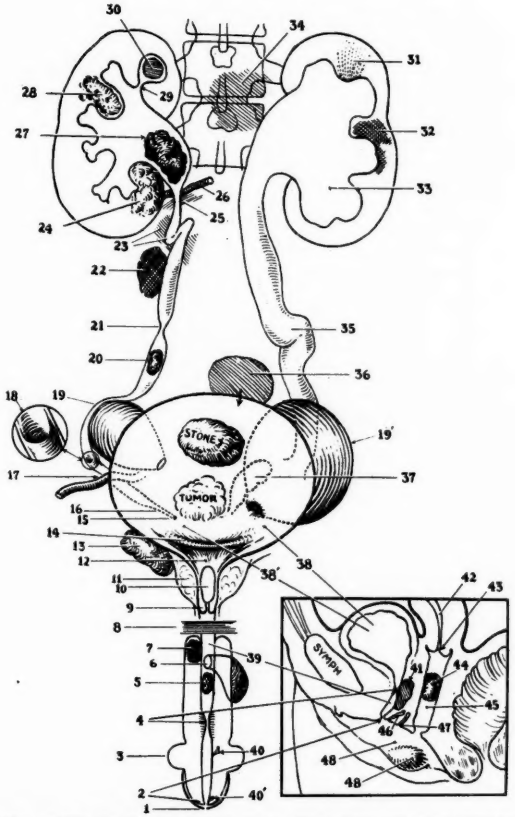

Fig. 1.—Direct and indirect causes of pyuria. These lesions, the clinical manifestations of which so commonly lead to the inadequate diagnosis pyelitis, either acute or chronic, can be identified only by a thorough urologic examination. *1*, Stenosis of prepuce; *2*, stenosis of urethral meatus; *3*, paraphimosis; *4*, urethral stricture; *5*, urethral stone; *6*, urethral diverticulum; *7*, periurethritis; periurethral abscess; *8*, cowperitis; chronic external sphincterospasm; *9*, congenital valves of posterior urethra; *10*, hypertrophy of verumontanum; verumontanitis; *11*, prostatitis; prostatic abscess; *12*, contracted bladder neck; median bar; *13*, periprostatitis or pelvic suppuration; *14*, mucosal fold at bladder outlet; trigonal curtain; *15*, stricture of ureteral meatus; ureterocele; *16*, ureterovesical junction stricture; *17*, vascular obstruction of lower ureter; *18*, congenital ureteral values; *19*, ureteral obstruction by diverticulum compression; *19*,[1] diverticulum; *20*, ureteral stone; *21*, ureteral stricture; *22*, periureteritis; periureteral phlegmon or abscess; *23*, ureteral kink; periureteral fibrous bands; *24*, renal tumor; *25*, ureteropelvic junction stricture; *26*, aberrant vessel obstruction of upper ureter; *27*, pelvic stone; *28*, renal tuberculosis; *29*, stricture of calyceal outlet; *30*, calyceal stone; *31*, pyelonephritis; *32*, pyonephrosis; *33*, "pyelitis"; infected hydronephrosis; *34*, perirenal suppuration invading urinary tract; spinal disease (Pott's, etc.); *35*, hydroureter; *36*, pericystic abscess rupturing into bladder; *37*, seminal vesiculitis; *38*, neuromuscular vesical disease; *38*,[1] cystitis; *39*, urethritis; *40*, folliculitis (Littre); *40*,[1] folliculitis (Morgagni); *41*, periurethritis; periurethral abscess; *42*, endometritis; *43*, cervicitis; *44*, foreign body in vagina; *45*, vaginitis; *46*, skenitis; *47*, folliculitis of introitus; *48*, bartholinitis.

quires cystourethroscopy, ureteral catheterization and often pyelography.

HEMATURIA

The usual causes of hematuria are shown in Fig. 2, but blood in the urine may be due to practically any urologic lesion from the prepuce to the renal tubules. Twenty to twenty-five per cent of all patients with urologic disease pass blood in the urine, often microscopic. In ninety-five to ninety-eight per cent of these cases the hematuria arises in the urinary tract itself. In three-fourths of these cases the hematuria, persistent or intermittent and gross or microscopic, is due to tumor, infection, calculus, or nephritis. In forty per cent of urologic neoplastic disease there is hematuria. In ninety to ninety-five per cent of renal tumors hematuria is produced and, under the most favorable therapeutic conditions, four to twenty per cent of these patients will be alive five years later. Eighty to ninety per cent of bladder tumors cause hematuria with a five-year-cure outlook of twenty to twenty-five per cent. All urethral tumors cause hematuria with a twenty-five to fifty per cent hope of cure. Eight to fifteen per cent of prostatic neoplasms produce hematuria with a likelihood of five-year-cure of not over one per cent. Ninety-five to ninety-eight per cent of urinary tract calculus disease produces hematuria, either gross or microscopic. Twenty-five to eighty per cent of bladder infection is accompanied by hematuria and thirty to fifty per cent of patients with nephritis have hematuria.

In addition to the causes of hematuria above indicated, this symptom is frequently found in certain general diseases such as malaria, rheumatic fever, scarlet fever; in chronic infections such as endocarditis, purpura, syphilis; in Hodgkins Disease, leukemia; in vitamin deficiencies, notably scurvy. It also is observed in urinary tract anomalies, particularly anomalies of renal fusion and malposition and notably also in polycystic disease (fifty per cent). Blood may

also appear in the urine consequent to disease adjacent to the urinary tract and invading the latter: acute appendicitis, salpingitis, intestinal tuberculosis, diverticulitis of the colon, osteomyelitis of the pubis and so forth. As a rule patients with these conditions are unlikely to present a problem to the Medical Director other than rejection. The hematuria following drug ingestion, particularly of methenamine, mandelic acid and sulphonamide compounds, phenol, salol and the like, may be temporarily disadvantageous to the insurance applicant. Hemoglobinuria is not to be confused with hematuria, nor is the dark red coloration of the urine observed in some persons following the ingestion of beets.

In the present discussion we are more seriously concerned with the problem of the patient whose urine shows minimal pyuria and/or hematuria. It is recognized that the lesions heretofore indicated may cause minimal as well as gross hematuria and therefore add to the complexity of the interpretative problem. At the outset it must be recognized that normally in a twelve-hour period up to 500,000 red blood cells and up to 1,000,000 or more white blood cells may appear in the urine (Addis). Yet this number is relatively so small that their recognition is unusual in the casual routine urinalysis.

At the risk of presenting certain strong personal convictions which you as Medical Directors of Insurance Companies may deem technically impracticable for mass life insurance examinations, let us briefly consider the manner of collection of the applicant's urine specimen. Urologists are daily examining large numbers of patients of both sexes referred with the diagnosis "pyelitis." These individuals are said to have this disease simply because the laboratory reported pus cells present in the voided urine specimens. Yet examination of the properly collected specimens commonly shows no abnormality. Especially is this so in the female and in my own work, both office and hospital, no time is spent in urinalysis of voided specimens from this sex; we insist that only

urine obtained by catheterization be subjected to examination. This applies to females from neonatal to advanced age. I am aware that the proportion of women applicants is much less than men but from now on they must necessarily be seriously considered if the present problem arises.

The method of urine collection by voiding as usually performed by females is scientifically so objectionable as to render the microscopic findings misleading to say the least. Occasionally a woman will wash the labia and introitus thoroughly before voiding and less often will separate the labia when voiding. Yet even with these conditions met there is still danger of contamination of the specimen by cellular debris which, even though only desquamated epithelium, will be reported as pus by at least fifty per cent of laboratory technicians. This statement is made most seriously and as a result of intensive personal study of this problem. Unless I know the specimen to have been properly collected, the only items in a submitted urinalysis report to which full credence is given are the specific gravity estimation and the glucose test. Yet on the basis of such a report, the patient is given an erroneous diagnosis.

Aseptic catheterization of the female with a soft rubber catheter eliminates these errors and only by catheterization of the female can a specimen be obtained for accurate bacteriologic study. Finally, the trauma of gentle aseptic catheterization with a soft rubber catheter inserted under visualization is infinitely less than that of scrubbing of the labia and introitus preparatory to the relatively "clean" collection of a voided specimen. It is appreciated that technical, professional and sometimes personal difficulties might possibly arise from insistence on catheterized specimens from all female insurance applicants. Yet the physicians examining insurance applicants in their respective communities are on the whole a superior group and should experience no difficulty in performing a prescribed catheterization technique. At any rate, and disregarding diabetics, the female declined insurance be-

cause of the urinalysis of a casually voided specimen is entitled to a re-examination of the urine — and this a specimen taken by catheterization.

Similarly, the urinalysis report of the specimen collected from the male by voiding through a prepuce, and especially when the preputial orifice is small, is equally as inaccurate, misleading and unfair as when the specimen is ordinarily voided by the female. The redundant prepuce notoriously harbors smegmatic and cellular debris. I strongly suspect that in many of the insured recorded in the previous paper and subsequently showing no evidence of urologic disease, the urine specimen was obtained by this usual and unscientific method. Yet a thoroughly satisfactory and accurate specimen may be collected from the male by voiding if the following procedure is employed. The prepuce is widely retracted, the glans and separated meatus are cleansed with soap and water or, if a bacteriologic examination is to be made, with an antiseptic solution such as bichloride or oxycyanide of mercury 1:1000. The man then voids into a clean receptacle. This specimen will probably contain a few urethral and prostatic shreds which may or may not be significant. If the specimen is to be bacteriologically examined, the male voids a few cubic centimeters before collecting the subsequently passed urine in a sterile vessel.

It seems to me that insistence on these advocated methods of urine collection should advisedly be absolute when the policy is substantial or when pus or blood is found in amounts considered significant. Certainly examination of a properly collected specimen will permit the most accurate evaluation of the individual as an insurance risk from the standpoint of the urinary tract.

Preferably, the collected specimen should be examined when fresh. The microscopic examination of long-standing or mailed-in specimens must be considered largely inaccurate, unless a satisfactory preservative such as the Wolff tablet is used. Although leukocytes are preserved in an acid urine, they read-

ily disintegrate in alkaline urine and their nucleoprotein will give a positive albumin reaction. Too commonly, desquamated epithelial cells and other urinary debris are reported as pus cells. Except for occasional mononuclears no cell should be called a pus cell unless the characteristic polymorphic lobate or "pawnbroker's" nucleus can be demonstrated. The microscopic examination of the pus cells gives no clue to their origin and the number, whether single or clumped, is of only relative concern. Serious urologic disease often exists when pyuria is negligible or absent.

Red blood cells are easily recognized in the fresh specimen but they tend to crenate and disintegrate rapidly upon standing—another reason for prompt examination of the freshly-voided specimen. Yet I have learned from Doctor Ungerleider that preservation of the specimen by the Wolff tablet satisfactorily preserves both red and white blood cells, as well as casts.

Parenthetically and in closing, it seems to me that insurance companies should subject applicants for substantial policies to an excretory urographic study in the same manner that chest roentgenography and electrocardiography are so generally employed. By this method many asymptomatic calculi, urinary obstructions or other uropathology will usually be revealed. By making a cystographic exposure after the patient has voided to his best at the end of the urographic study, the presence of vesical residual urine can be urographically demonstrated and estimated. Many bladder-neck obstructions are satisfactorily demonstrable by this method. Such observations are of particular interest and value in men over thirty-five years of age, and such studies could well be made an absolute requirement in all applicants showing minimal hematuria or pyuria when their condition otherwise would make them acceptable for ratable insurance. By this method urinary tract changes characterized by morphologic alterations are usually demonstrable; the interpretation of the urogram should be entrusted to the expert in the Home Office.

SUMMARY

The commoner causes of pyuria and hematuria have been discussed together with the relative prognosis.

Acceptable methods of urine collection have been described.

The employment of excretory urographic study is recommended as part of the examination for life insurance, particularly when substantial policies are sought.

Abnormal hematuria and/or pyuria always merit serious clinical consideration, even though the patient appears to be in excellent health. Frequently the cause of these abnormal urinary findings cannot be ascertained without cystourethroscopy, catheter examination and urographic study of the upper urinary tract.

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PRESIDENT WENSTRAND—The subject is open for general discussion. Dr. Harry E. Ungerleider!

DR. UNGERLEIDER—In connection with the recent revision of our Medical Underwriting Manual, we made some studies on so-called hematuria and pyuria on examination, and I will read a report made to Dr. Daley under date of January 24, 1940.

"Assembly of Material

We have abstracted from the file of substandard issues, 1925 through 1937, all policies falling within the following classifications:

(1) Cases rated chiefly for 633X or 634X either alone or in combination with other impairments not in the 601X–609X (Albumin) group. The 633X or 634X, if it was the sole loaded impairment, was loaded at least + 30; if there were other loaded impairments, the 633X or 634X was loaded at least as heavily as all the other impairments combined, and in no instance were the other impairments loaded sufficiently to have caused the issuance of a substandard policy in the absence of the 633X or 634X.

(2) Cases rated + 30 or more for either 633X or 634X combined with one of the albumin codes 601X–609X, no other impairments being present which would have caused the issuance of a substandard policy in the absence of the combination urinary impairment being considered. The relative loadings applied to the 633X, 634X or 601X–609X were disregarded. In some instances the heaviest loading was given to the hematuria or pyuria, in some the loading was divided equally, but in most instances the albumin was rated more heavily.

"*Method of Analysis*

The policies were observed to their 1939 anniversaries with the expected claims derived from the E. L. A. S. 1925–1937 Issues Table extended one year. Four separate groups were studied:

(1) 633X, Hematuria on examination

(2) 634X, Pyuria on examination

(3) 633X with 601X–609X, Hematuria with albumin, both on examination

(4) 634X with 601X–609X, Pyuria with albumin, both on examination

	Entrants	Claims	Expected	Mortality Ratio	Average Rating of Entrants
Hematuria alone	218	3	7.96	38 ± 24%	+51
Pyuria alone	504	28	20.97	134 ± 17%	+55
Hematuria with albumin	280	21	8.45	249 ± 36%	+74
Pyuria with albumin	217	13	5.82	223 ± 41%	+76

"In no instance was there any abnormal number of persons dying with more than one claim on the life.

Breaking the groups into divisions by age at issue revealed no significant contrasts within the groups, a condition rather to be expected where the volume of material is so small.

"The causes of death, however, revealed sharply the pathological significance of the combination impairments. Of the 27 lives represented by the 31 claims in hematuria or pyuria alone, only 2 died as a result of urinary conditions. Among the 30 lives dying in the combination group, 13 died of conditions relating to the genito-urinary system. The mere addition of the ratings for albumin and hematuria or pyuria appears to be quite inadequate to provide for the extra mortality when both abnormalities are present."

PRESIDENT WENSTRAND—Are there any other comments or questions?

DR. ROBERT M. DALEY—Dr. Wenstrand, I think it might be well here to bring to light that which is in all our minds, and that is, that these classes of hematuria and pyuria cases that are insured have the history of any disturbance more or less current, eliminated. These are supposed to be healthy people without any symptoms, who apply for insurance and in whom these abnormal conditions are found.

In Dr. Campbell's case and from the clinical point of view, and not alone his cases but also in regard to some of the classes to which he referred, they are collected by

practitioners in urology, by clinicians. These people have come to the clinics because they are sick with some disturbance which points to the genito-urinary tract, and as Dr. Campbell mentioned, pain is one of the most common symptoms; so that this will account to a certain extent for the discrepancy which appears and why it would seem that the life insurance experience is so favorable.

Most of the insurance companies now have their urinalyses made at the home office by competent physicians or by competent technical people. They could not afford to do otherwise, because I estimate in the experience of the Equitable that our laboratory pays us for what it doesn't find, pays for its own upkeep in what it does not find.

Cases of albuminuria which are discovered by the examiner are sent to the laboratory and we find that it is due to something else, and so we must be very careful that we do not find something in the specimen that isn't there.

Not long ago, I caused about two hundred cases with minimal hematuria to be sedimented, and the sediment examined for tubercular bacilli. I had a hunch that we might find a tubercular kidney in them. Well, we found one case, one specimen that had an acid-fast bacillus in it, and we were not sure then that it was not smegma bacillus, and so I had to give up the study because of our inability to follow up such cases, a condition which does not ordinarily exist with the clinician because he has a better chance to follow them up.

PRESIDENT WENSTRAND—Any furthur comments or questions?

DR. ROBERT L. ROWLEY—I would like to ask Dr. Campbell a question. He made a distinction between white blood cells and pus cells. I gather from Dr. Tegtmeyer's paper that his study was made on white blood cells; in other words, what insurance companies usually call pus cells or white blood cells. But Dr. Campbell specified a particular kind of a white blood cell, a polymorphonuclear or leukocyte, as the kind of blood cell he was talking about. I think that point might be made clearer.

I would also like to ask Dr. Campbell what he means by minimal pus and blood in the urine. I would also like to ask him if he could tell us a little bit about the difference between pyelitis and pyelonephritis.

PRESIDENT WENSTRAND—Are there any further questions? I appreciate very much that Dr. Campbell spoke about the correct way of collecting specimens of urine. We know only too well that that would be eminently desirable, but in life insurance examinations, it becomes almost impossible to do that except in the doubtful cases, where we do want to be sure that we are not getting, especially in the male, the sediment from the urethra. But it is fine to know that we have a method whereby we probably can exclude some of the abnormalities which are found in our home office specimens.

If there are no other questions, I shall ask Dr. Campbell to close and answer the questions which have been asked.

DR. CAMPBELL—In regard to the first question, regarding the identity or identification of pus cells, I am sorry it was so confusing. I did not make myself clear. There are a few mononuclears that get into the urine which technically are leukocytes, but the cells that we consider pus cells are the lobate, pawnbroker's nucleus type of polymorph. On staining, you will find they are practically all neutrophil cells, and those are the cells that we call pus cells.

The number of pus cells in the urine is of only relative concern. It does not indicate the severity of the disease, except in the most general way. The method of identification that we employ is the examination of the urine centrifuge, the freshly shaken urine specimen. If we have more than two or three leukocytes in the high-power field, we consider that the patient is entitled to clinical attention. If we find that many blood cells, or that much blood, we feel the same way about it. There are others who feel that up to five or six in the high-power field, of similar specimens, is within normal limits, and it may well be. But certainly the persistency of

that much blood or blood over that amount entitles a patient to clinical investigation, we believe.

On the question of pyelitis, I particularly stressed that in speaking of it at the time, saying that we put it in quotes, because although we realize that pyelitis is a term that has been used for a long time to signify renal infection, it is used so loosely that it really has no pathological significance. Pyelitis, strictly speaking, means inflammation of the kidney pelvis, and four out of five doctors will give you that definition if you ask them what is pyelitis.

On a pathological basis, the inflammation in the kidney pelvis itself can be wholly disregarded. It is a matter of little or no significance. The important lesion is in the renal parenchyma, and histologically, in 98 per cent of the cases at least of renal infection, it is an interstitial, suppurative nephritis, with the invasion of the pelvis from a hematogenous infection; that is, with cortical and medullary involvement, first and secondary involvement of the kidney pelvis, you have an anatomic pyelitis, but the pathologic picture is pyelonephritis, and the same way with an initial infection of the pelvis, which has never been proved in the human. It has been demonstrated histologically in animal experiments, but never after forty-eight hours—or the involvement of the pelvis, a secondary invasion of the renal parenchyma—and we insist on the term "pyelonephritis" if you are going to be pathologically correct and say what you mean.

I think that was all.

PRESIDENT WENSTRAND—Did you have something else to say?

DR. ROWLEY—I just wanted to ask Dr. Campbell about the collection of the split specimen from microscopic examination; was that recommended by Dr. Campbell? Do you think that specimens from microscopical examinations, from the male at least, or from both, should be split specimens and the second half only counted as being of pathological significance?

DR. CAMPBELL—I think it is a fair test, and certainly you should use the second specimen for bacteriological culture.

PRESIDENT WENSTRAND—We now come, if I may say so, to something that might be called an interlude. Our very good friend from Canada, Lieutenant-Colonel Keith Gordon, spent some fifteen months or more in Britain during 1940 and early 1941—or was it early in 1942? Well, at any rate, he will tell you that. I feel that it is particularly fortunate that he will tell us some of his experiences.

I want you to know that Dr. Gordon is a Canadian dating back three or four generations, and he came from western Canada, so that we may refer to him as a Westerner. Dr. Gordon!

EXPERIENCES WITH THE CANADIAN ARMY IN ENGLAND

By Lieutenant-Colonel J. Keith Gordon

In July 1940 I had the honour and good fortune to proceed overseas as an officer on the staff of No. 1 Canadian General Hospital. This unit, which was designed to accommodate six hundred bed patients, was mobilized in the city of Montreal and recruited its officer and nursing sister personnel from the teaching hospitals affiliated with McGill University. The voyage of our convoy of transports was both pleasant and uneventful, except for some brief moments of anxiety, and when we disembarked in the United Kingdom we proceeded to our destination.

The barracks in which we were temporarily lodged was built shortly after the Peninsular War and bore the name of one of the great battles of that War. The quarters were proverbially cold and dreary, but we concluded that since they had housed some of the greatest heroes of Britain's wars, they were probably good enough for us. I obtained some consolation in exploring the possibilty that even the bed in which I slept had been occupied by the officer who led the Charge of the Light Brigade, or the great Lord Kitchener. Whoever the poor chap was, I can only say that he must have found it damnably uncomfortable.

In our moments of leisure we visted many places of interest. To-day it is trying to the soul to observe the destruction which the Nazis have produced in this country. In an apparent attempt to bomb Windsor Castle they have repeatedly over-shot the mark and, as a result, have made a shambles of a famous boys' school. It is equally likely that Field-Marshal Goering decided that it was expedient to exterminate the little boys who played there. If this be true, his plan has been at least partly successful for in two separate attacks the shooting at the school has been remarkably accurate. (I

might say that there are no military targets either in or about Windsor.) In respect of Winchester Cathedral, there is a legend which is firmly believed by the inhabitants of the town and it is to the effect that Hitler, in anticipation of an early conquest of England, has orderded that no bombs be dropped in its vicinity, so that it may be preserved as an appropriate place for his coronation as King-Emperor.

The defensive role assigned to the Canadian Army has been a keen disappointment to them. Expecting early action, they became envious of the Empire troops who were sharing battle honours with those of the Mother Country in other theatres of war, while they were forced into inaction by a change of strategy on the part of the enemy. Only through the employment of a rigorous syllabus of training and the fact that the element of surprise has to date rested almost entirely in the hands of our opponents, have the Canadians been delivered from a wholesale "browning off," which is a term used in the forces to describe a state of boredom fringing on insanity. But with the arrival in Britain of increasingly large numbers of troops from the United States, our men and the Home Guard have been given some relief in the matter of the invasion watch. Recently they have turned to the offensive and their hopes and morale have been greatly bolstered by the knowledge that they will soon fight their way into the very heart of Germany, in company with the great army that is being trained in this country. I cannot refrain from recounting the expression of opinion that was voiced in our mess when we received by radio the astonishing news that Japanese planes had attacked Pearl Harbour. It was: "They have awakened a sleeping giant."

We arrived in England at a time when the country was suffering from the painful effects of a great military defeat. The remnant of the British Expeditionary Force had just been evacuated from France, with the loss of its entire equipment, and feverish attempts were being made to equip Britain's new and expanding army and to provide planes for the Royal Air Force. The Home Guard, which consists of vet-

erans of the last war who are over-age, men who are unfit for general service, and boys who have not yet reached the call-up age, and which is now a well-trained and well-equipped force, had not as yet been organized, and more through instinct than good management the men of the English countryside took up their guns and sporting rifles and undertook a little hunting of parachutists and spies on their own account. With the whole of the coast of Europe in the hands of the enemy, the British were facing the grim prospect of an invasion of their country and Mr. Churchill not only gave an indication that it was imminent and that the landing of a hostile force could not be entirely prevented, but he also warned of how the enemy would be received, when he said: "We shall defend our island whatever the cost may be. We shall fight on the beaches, we shall fight on the landing grounds, we shall fight in the fields and in the streets, and we shall fight in the hills; we shall never surrender."

Up to this time there had been relatively little bombing of England. Evidence of damage from air attack was so slight and scattered that it was scarcely visible. For the first two weeks that we were in England we did not even hear the siren. Suddenly, however, alerts were being sounded at intervals throughout the day and night. Large formations of enemy planes appeared overhead and, although we were slow to appreciate it, a crucial battle of the War had begun — the Battle of Britain. As you all know, the Battle of Britain was a struggle for the control of the air over Britain, without which no invasion attempt could be successful. The enemy sought to destroy the fighter strength of the numerically inferior R. A. F. and to render the latter's air-fields unserviceable. The R. A. F. successfully met the challenge by refusing to engage the enemy with a large force of fighters at any one time, to avoid the risk of a crippling blow, and relied more on the skill and daring of its personnel seated in a very fast and easily-maneuverable plane. The Battle of Britain may have been said to have culminated on September 15th, 1940, on which date 152 boys in the R. A. F. fought the enemy from

dawn to dusk without respite and succeeded in destroying 185 German planes over the British Isles. In view of the reputed strength of the Luftwaffe at that time, this prodigious loss in planes might have been sustained for a short period if the desired result were to be accomplished, but the fact that there was an abrupt change in the enemy's strategy at this point to the bombing of the larger cities by night, suggests that the morale of the Luftwaffe had been so severely damaged that Goering feared the risk of suffering further defeats of a similar nature.

The spectacle of the wreckage of destroyed enemy planes strewn over the length and breadth of the country, was a tonic to the English people, for it meant that the invasion of their country had been postponed, if not abandoned for all time. What we of the United Nations owe to a body of very gallant youths who fought the Battle of Britain and to R. J. Mitchell, the designer of the Spitfire, history will record.

In October 1940 our unit received orders to take over another hospital. The hospital consisted of a block of huts, each hut having accommodation for 40 patients, and it had been built and equipped by the Ministry of Health according to a standard plan. Some 175 similar units had been constructed in various parts of the United Kingdom, in anticipation of heavy air-raid casualties and they gave to the already existing hospitals an added potential of about 100,000 beds.

During the so-called "phoney war" period, many plans had to be formulated without the benefit of past performances, for there was a new technique in warfare. Various estimates were made of civilian casualties resulting from attack from the air, and one observer predicted that a "first-class air raid" might result in 50,000 dead and 300,000 wounded. Everyone imagined that the war would begin with immediate mass raids on London and few had forgotten Lord Baldwin's uncomfortable phrase: "The bomber will always get through." Incidentally, the total number of air-raid casualties in the United Kingdom from the beginning of the War to October 31st, 1941 have been 43,113 killed and 53,813 wounded, which are sur-

prisingly low figures in view of the intensity and extent of the attacks. It is of interest to note that the number wounded exceeded the killed by merely 10%, which indicated that mortuaries are almost as desirable as hospitals when the civilian population is attacked from the air in a large scale manner.

How did British medicine meet the challenge of September 3d, 1939 and what was the machinery that put the medical services in motion? With remarkable foresight the British Medical Association began as early as 1937 to compile a register of every doctor in the country and to classify them according to their aptitude for service in the Army, Navy, Air Force or the Home Front. A Central Emergency Committee and Local Emergency Committee were formed, so that when war broke out the Committees were in a position to give immediate advice to the Ministry of Health. There was very little difficulty encountered in allocating doctors to the armed forces, but the matter of providing personnel to look after air-raid casualties was somewhat of a problem, for it involved such matters as duty roster, pay, the role of the specialist in the larger cities, and the ordinary needs of the civilian population. Finally, before the end of 1939, with the formation of the E.M.S., or Emergency Medical Service, the medical profession and the hospitals of Great Britain became nationalized for the duration of the war. The E.M.S. reported to the Ministry of Health. Its brain-trust consisted of "special advisers", drawn from the British Medical Association and the National Research Council. (Geographically it functioned on the basis of the eleven divisions into which England and Wales were divided for purposes of national defense.) The key men were a so-called hospital officer in each region, assisted by the county medical officer and county borough medical officers of that region, while, in addition, in the larger centres of population, a group officer supervised staffing, treatment and the matter of hospitals that could be set aside for specialized work as, for instance, plastic surgery.

When the expected air-raid casualties did not materialize some of the Ministry of Health emergency hospitals were

turned over to the armed forces, including the one to which we were posted. The majority of them have been placed in close proximity to a sanatorium, poor-house, or mental institution, in order to obtain good land on a low rental basis and to make use of the already existing arrangements for obtaining power, water and sewage disposal. Such institutions are usually well situated in respect of main roads and railways, as well as being in the quietest and most beautiful spots of rural England. For reasons that I am not at liberty to divulge, the location of our hospital was not an ideal one, in fact it turned out, as we expected to be a rather hot spot. However, the emergency well of the neighboring mental institution proved to be a great boon when our water supply was cut off by enemy action for periods of as long as three weeks. The supply of electricity and gas usually followed the example of the water, at which times an emergency coke oven took the place of the gas burners and a small auxiliary power unit serviced the operating room and x-ray department, while hurricane lanterns lit the wards.

From the very moment that we arrived we were treated to nightly air-raids and an occasional daylight raid by single planes or small formations. High explosive bombs frequently dropped within a few hundred yards of our hospital without causing any damage or casualties, and we were actually hit by incendiary bombs, following which we became very fire conscious and took steps to see that we were adequately protected, particularly in the matter of a generous supply of static water. By static water is meant small pools or reservoirs, either natural or artificial, that are independent of the main supply. The British have placed static water in every possible location, even utilizing the cellars of blitzed buildings after removal of the debris, and have manufactured very large quantities of portable pumps operated by two cylinder motors to cope with the fire hazard. As for the volunteer fire services, their work has been superb, both in the matter of skill and bravery. It is the technique of night raiders to first start fires and later shoot at them with high explosive, a com-

bination which puts great demands upon the fortitude of the fire-fighters. An example of their endurance is shown by one of the casualties which we received, whose left arm had been, to all intents and purposes, amputated by a bomb fragment and most of the gluteal muscles on the same side removed, and who continued to direct the stream from the hose that he held for fear that he might otherwise harm some of his comrades.

On the night of November 19th, 1940 the Luftwaffe attacked the little city of Coventry in force. The raid began shortly after 9 P.M. and continued until about 3 A.M. the following day. A full moon made the visibility excellent. Great damage was done to the city and many civilians were killed and wounded. From the speed with which the enemy flew from the city after unloading his bombs and the low altitude at which he was flying, we gathered that he was hurrying back to reload for a second run, which was quite probable in view of the intensity and length of the attack. He had very little opposition in this raid, for our night defenses were not nearly so formidable as they are today.

Immediately following the attack on Coventry the authorities evacuated to us the bed patients of two large children's hospitals which were located in extremely vulnerable spots. There were some complicating factors associated with the admission of 300 children ranging in age from one day to twelve years, since we possessed neither cots, feeding bottles, nor diapers, and our nursing sisters had not yet arrived from Canada; but from the British and Canadian Red Cross we obtained appropriate equipment and with reasonable efficiency we operated a children's hospital for a period of about two months. The cases were both medical and surgical, a few of the latter being air-raid victims. The newly-born baby was a healthy girl whose home had been blitzed a few hours after her birth. Fancy suffering the tribulation of being born and bombed all in one day!

With few exceptions the children tolerated the air-raids well. Some of the cases of chorea seemed to become more

jumpy, but the great majority slept soundly at night in spite of the noise and vibration. This lack of fear on the part of the children has been a general observation and it is a criterion of the example set by their elders and the quality of the discipline that exists in their homes. Even the dogs of jittery families whine and make a great fuss when they hear the siren and only become mollified when allowed to proceed to the Anderson shelter.

The enemy continued to raid England almost nightly until shortly before Christmas of that year. In one city alone over 100,000 homes were made uninhabitable. Glass cannot be replaced in the United Kingdom to-day, so that most of the windows have remained boarded up. The fact that at this point he abruptly cut short his visits suggested to me that his underground channels of communication are not quite so efficent as we are generally led to believe that they are. The Ministry of Health was quick to realize the grave dangers associated with a water shortage and had placards posted in every available spot, bearing the words: "Boil all water. Boil all milk," while other posters and cars carrying loud speakers described how and where free inoculation against typhoid fever could be obtained.

In the period following these heavy raids and the last batch of children had been discharged, we began to admit members of the armed forces exclusively until the Thursday night before Easter, 1941, when the enemy again attacked the city of Coventry in force. Few of us slept at all either that night or the following one, for 87 high explosive bombs dropped in the hospital area, many of them being of the delayed action variety, and a large number of civilian casualties were admitted. Prior to the raid we had received a wounded German airman and it is of interest to note that he seemed to dislike the bombing even more than the other patients during that rather disagreeable night. We concluded that although he had dropped many bombs he had never been on the receiving end. How times have changed!

By this date we had learned from our British confreres the need of a resuscitation ward and the value of what is termed "tirage". A resuscitation ward is one which is set aside for the treatment of shock and "tirage" is a French word which when freely translated means "the selection of risks," a matter of equal importance to the proper disposal and mortality rate of a large group of air-raid casualties as it is to the mortality rate of a life insurance company. A physician with some knowledge of surgery and an abundance of that indefinable quality of mind which is often described as "good judgment" is usually assigned to this post and it is his responsibility to allot the cases on admission either to the resuscitation ward, to a surgical ward, or immediately to the operating room. It was our experience that all air-raid casualties suffer from some degree of shock, often out of all proportion to the extent of the injuries received, and it is of primary importance to treat shock first and foremost and to delay all surgical procedures until such time as the patient has recovered from shock. In our resuscitation ward we used quantities of plasma and glucose saline, reserving whole blood for patients suffering from blood loss. The application of warmth to the extremities and the administration of hot drinks were the only additional forms of treatment. We found hot tea to be an excellent beverage since it combines fluid, sugar and warmth and is well tolerated. Extensive wounds were treated by surgical debridement and Trueta's closed plaster method with good results.

We also began to appreciate the value and convenience of the location of our X-ray Department, which had been placed in a section adjoining the operating room, since the matter of the location of foreign bodies is so important in this type of surgery. Some of our fatal cases developed gas gangrene as a result of the presence of foreign bodies which were non-opaque to the x-rays.

At this point I would like to comment on the teaching and practice of first-aid to the injured. I believe I can say that it has been the general experience in the United Kingdom

that while the St. Johns Ambulance Corps and the Red Cross Society have performed a very fine service in the past in the matter of teaching first-aid, their methods do not meet the requirements of the present emergency. Too much is taught about too many things in too short a time with the result that the student becomes either completely bewildered or he feels obliged to put into practice the elaborate dressings and splints that he has seen described in the first-aid manuals, the net result being delay and unnecessary handling of the patient, both of which are decidedly harmful. The first-aid worker should be thoroughly trained in three fundamental principles only—the control of haemorrhage, the recognition and first-aid treatment of shock, and artificial respiration, and he should receive his instruction from surgeons who are constantly dealing with accident cases, and not from laymen or retired physicians.

Medical, as opposed to surgical, cases admitted as a direct result of air-raids were few in number. There were some instances of hysterical mutism and we saw a small number of cases of so-called "crush syndrome", in which a fatal anuria follows multiple crushing injuries. We did not see any recognized cases of blast effect on the lungs.

So far as patients from the armed forces were concerned, the largest single group was made up of infections of the respiratory tract, of which the majority were atypical pneumonias, probably of virus origin. Pneumococcus or lobar pneumonia, on the other hand, was relatively rare. Patients suffering from atypical pneumonia when reviewed as a group had fairly characteristic features, the outstanding ones being hacking cough with mucoid sputum, absence of pneumococci and the presence of a mixed flora in the sputum, relatively few physical signs, usually out of all proportion to the x-ray findings, transient opacities in the x-ray film which are not unlike the lesions seen in tuberculosis, a low or normal white cell count, and the lack of response to sulphonamides. The disease is highly contagious and it is worth noting that most

of the epidemics described in the literature have occurred in college students or in garrisoned troops.

The next largest group was the gastro-intestinal one, with peptic ulcer running only slightly behind functional disease. The high incidence of peptic ulcer is undoubtedly due to the fact that if the recruit on enlistment wishes to conceal a previous attack there is no practical method of discovering it.

As was to be expected in a relatively young and selected age group, the balance consisted largely of anxiety states, including effort syndrome, and acute infections such as rheumatic fever and cerebro-spinal fever. Diseases of the skin formed a fairly large group, in which connection it is worth noting that pediculi were conspicuous by their absence, and while scabies is still fairly common it is now curable in twenty-four hours by the benzyl benzoate treatment.

The degenerative diseases were rare, and the paucity of cases of pulmonary tuberculosis and organic disease of the heart pay tribute to the value of mass x-rays of the chest on enlistment as practiced in the Canadian Army.

In the matter of anxiety states, of which there were a considerable number, I cannot help making some reference to the part which the women at home can play in the prevention of this serious malady in troops abroad. A great many of the patients whom we saw that were suffering from functional nervous disease admitted that they had been greatly worried and upset by news received from their wives or sweethearts. Mournful communications from home may cause more damage of the morale of troups abroad than anything which the enemy has to offer and it is my opinon that appropriate steps ought to be taken to deal with this problem through the medium of the various women's organizations.

We were fairly well supplied with the basic drugs. Liquid paraffin was never at any time procurable and we were forced to practice the strictest economy in the use of some substances such as codeine. On one occasion when a large consignment of drugs was destroyed by enemy action we were suddenly

confronted with a complete absence of sulphonamides, at which time one of our officers, who was in charge of the venereal service and whose patients were consuming large quantities of these drugs daily, sent a dispatch to the quartermaster dispenser marked "Urgent". It read as follows: "No sulphanilamide, no sulphapyridine, no sulphathiazol, no potpermang. Please send Christian Science practitioner," to which the quartermaster dispenser humorously replied: "In lieu of above am sending copy of New Testament, Army Edition."

In spite of the shortage of food, the shelter problem, and the presence of constant danger, the general state of health of the inhabitants of these Islands, now in their fourth year of war and third under fire, is excellent. With 23,500,000 of their 33,000,000 people between the ages of 14 and 65 mobilized for full-time national service in some form or other—the remainder consisting of part-time volunteers, if they are not invalids—hard work and frugality have supplanted leisure and pleasure without deleterious effect upon the national health. There is a great shortage of meat, eggs, and fresh fruit. The civilian meat ration amounts to about the equivalent of two and one-half chops per week. The egg ration is one egg every two weeks. What fresh fruit reaches the country is reserved for school children. And yet the people of the United Kingdom are on the average better fed than they have ever been before for in the first time in their history they are being fed scientifically. In the absence of butter the Ministry of Food has decreed that all margarine shall contain adequate amounts of Vitamin "A" and "D". Vitamin "B" and calcium are supplied in the so-called "National Loaf", while Vitamin "C" is present in potatoes and other vegetables in quantities that are considered sufficient to maintain health. With civilian cars off the roads entirely, the number of road accidents due to the black-out have dropped to an insignificant figure. The black-out is depressing, particularly in the winter months, but no one complains about it when the alternating motors of the German bombers are heard overhead.

A problem which was fraught with great danger arose when England was being bombed almost every night from black-out to dawn for a period of about three months and thousands of men, women and children spent their nights in the city's subway system "bringing with them, in addition to their rags, their bags and their bedding, their coughs and their colds, their lice and their fleas, and their own manners and morals—spreading their belongings over the platforms, the passages and the stairways. Those who saw and smelt the result were appalled by the unsanitary state of affairs that prevailed and the risks to health and morale that were made imminent. Light, heat, ventilation, lavatory accommodation, a place to sit or even sleep on—these were almost non-existent."(1) The Government could not drive the people out of the subways, so that it had to face the problem of making them fit for habitation and as sanitary as possible. As a result of experiments that were conducted in an effort to sterilize the air and cut down droplet infection, two interesting observations were made; one was that the burning of cardboard diminished the bacterial content of the air and that the spraying of finely divided particles of a solution of hypochlorite, which also acts as a good deodorant, rendered the air practically sterile. The fact remains that either by good luck or good management there have been no major epidemics of disease.

The British people have a strange mixture of qualities which we who live on this continent find difficult to comprehend. They exhibit bravery and humour under extremely trying circumstances, yet their reticence, combined with a tendency to make little of themselves, creates in those who do not understand them the impression that they are a stupid race. They are only stupid in one respect, and that is they do not know the meaning of the word "propaganda". Apropos of this I would like to relate a tribute which was paid to them by one of the enemy and of which I had personal experience. On my return voyage from England I was appointed senior medical officer of our troop-ship which, in

addition to other details, carried German prisoners of war bound for prison camps in Canada. A part of my duty was daily attendance at an inspection of their quarters, the holding of their sick parade, and the care of the prisoners who were confined to the sick-bay. I had a good opportunity of viewing them at close quarters. The majority of them were of warrant officer rank and almost equally divided between the Air Force and the Navy. I would say that they were a good sample of the fanatical Hitler-trained youth. They were all fine physical specimens and their discipline and cleanliness under rather trying conditions was superb, in fact, rather disappointingly so. They were well clad in uniforms made of excellent material which, with ample supplies of chocolate and tobacco, had been sent to them through the medium of the German Red Cross. Some of the prisoners were survivors of the battleship Bismarck, and one of these who spoke good English told me that the Royal Navy had performed a rather remarkable feat in destroying what was thought to be an "unsinkable" ship. He said, "You English think that you are a lot of damn fools, but you are not such damn fools as you think you are."

It is scarcely necessary for me to draw attention to the fact that the United Nations have had little to brag about in the matter of accomplishments on the field of battle in this war. It is just as unnecessary for me to comment on the reasons for this. There may be some of us who at this time are beginning to entertain doubts and fears as to what the final outcome may be; but there can not be many who think in this way, for with our interest centered on life insurance medicine we must posses a profound respect for history.

Ian Hay(2) has written a narrative based on Lord Gort's Dispatches—both of which I have in my possession—describing the tragic experiences of the British Expeditionary Force in France from September, 1939 to the final evacuation from Dunkirk on June 3d, 1940. I believe he expresses the feeling of every one of us when in his introduction he says: "The day of victory is not yet, and may not fall for some time; for

we have learned a lesson, and that is that until we have consolidated our command of the air and endowed our young and spirited armies with the mechanised aid that they require, the decisive blow can not be struck. So we must wait, with fortitude and patience until, by the unremitting effort of the men and women of the factory and forge . . . . . these vital necessities are forthcoming and we can put forth our full strength. When that victory is won, but not till then, the Flanders campaign of 1940 (and I would like to add the Battle of the Phillipines) will fall into its true perspective—of as gallant a contribution to ultimate victory as Gallipoli or Corunna."

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PRESIDENT WENSTRAND—I know I do not have to tell you Colonel Gordon, how we feel about the splendid talk you have given us, but we do thank you from the bottom of our hearts.

Dr. Edward Adam Strecker, our last speaker, is a native of Philadelphia and there he was educated. To give an account of his many degrees, society affiliations and positions of trust could not add to his renown, so I shall simply remind you that he was a Major in the last World War, serving as Division Neurologist and Psychiatrist of the 28th Division both here and in France; also that Dr. Strecker is now Professor and Head of the Department of Psychiatry at the University of Pennsylvania Medical School and author of many books and articles dealing with all phases of nervous and mental disorders. Those of you who have not read "Alcohol—One Man's Meat" have missed something. Dr. Strecker from his wide experience will address us on "Military Neuropsychiatric Disabilities and Their Treatment." Dr. Strecker!

MILITARY NEUROPSYCHIATRIC DISABILITIES AND THEIR TREATMENT

By Edward Adam Strecker, M. D.

I should like to say that I am a firm believer in insurance, and as I look back over the last twenty years, it seems to me that it consisted of episodes of my worrying about getting together the premiums to pay for the insurance, followed by episodes of relief that I had succeeded in getting enough money together to pay them.

I suppose it is a kind of principle of insurance that there comes a time when it is too late, or almost too late, to take out insurance. Certainly, if one waits too long, the premium becomes exceedingly high. I wonder if it is not fair to draw an analogy between that and the present World War in which we are engaged. It is conceivable that we waited too long to take out our insurance against war, and that when we did take it out, we were willing to pay only a very small premium. Therefore the kind of insurance that we could buy for what we were willing to pay has furnished very inadequate protection. Then, when we finally had to take it, the premium we had to pay, of course, as in life insurance, was exceedingly high, and it is a premium that we are now paying, in the words of Winston Churchill, in blood, sweat and tears.

I suppose a war has two objectives—an immediate objective and a remote objective, which varies, sometimes the remote objective is good and sometimes it is bad. I think we all agree that the remote objective of this war is exceedingly good, to preserve not only democracy but also to prevent the world from falling back into barbarism. I know of no war that had a better remote objective; a more noble ultimate ideal.

Whether the cause is good, bad or indifferent, the immediate objective of war is always the same, and that immediate objective is *to kill.* Therefore, the immediate objective

of war, obviously, remains the same. This is true whether you are thinking in terms of a little war such as the cavemen had, with clubs or rocks, or whether you are thinking in terms of the great modern conflict in which we are now engaged.

Caveman even though he fought with the greatest enthusiasm could do comparatively little damage, managing only to cave in the few skulls of those who got close enough to his rock or club. Contrast that to modern warfare. I think it is perhaps even correct to say that there has been a very considerable change in the technique and method of war as compared to the thing that we used to call World War No. 1, but which now seems to me to be such a small and immature affair that perhaps it would be better to call it World Brawl No. 1.

From the little I know of military tactics and methods, which is a little less than nothing, it seems to me there has been quite a change. This war is outstandingly a gas engine war. It is predicted, and I think probably true, that artillery is on its last legs. It no longer has the place that it had in clearing the ground for the charge of the infantry. That will be done, and is being done by the RAF and other airplane services.

I remember, in World War No. 1, we had our first view of tanks in action. They were little machines like toys with two men in each one. As I look at the modern tank, it seems to me that one of those little gadgets could be pushed over with two hands, or bitten in half by a Russian soldier. Furthermore, I remember engaging in many bets in France—and the odds were very, very heavy and finally I couldn't get a bet at all—long odds that an antiaircraft gun would never hit an airplane. I never saw one hit. Now, they do it right along in very perfect style.

One thing has not changed much: The man who fights the wars. Unfortunately, or perhaps fortunately, I don't know which, men have not changed in the direction of being less

vulnerable to war from the standpoint of their nervous systems. I think they are more vulnerable. In fact, I think I can say I know they are more vulnerable.

One is not permitted to give restricted information, but I think one is permitted to say that thus far—I am talking now, not about the civilian population, but about soldiers—there is not the slightest reason to think or to hope that there will be any lesser degree of vulnerability of the nervous system than there was in the first World War.

It occurred to me, as I was waiting my turn to speak, that the opening of the section I wrote for SM–17, which is a medical manual of the United States Army for the present World War, might very well appeal to insurance men who are interested in disability insurance. There used to be disability insurance; there is'nt any more, I take it. But there used to be a great deal, and I happen to know that it was very much of a headache to insurance men. It seems to me that what is the opening sentence in SM–17 might very well appeal to men who are interested in disability insurance. This is it:

"Speaking of neuropsychiatric casualties, it is said that beginning with first aid, the objective of treatment is to return to duty as many men as possible, if necessary, with reclassification, and failing this objective, to send them back to civil life without neuropsychiatric handicaps incapacitating them for self-support."

How grand it would have been if you could have actually done that with disability insurance. I think you might have saved disability insurance instead of having to put it in the discard.

My advice to psychiatrists going into this war would be, first, to forget that they are scientific psychiatrists; that they are men who are capable and who like to work over diagnoses of patients to decide whether it is some kind of rare psychosis or not. There isn't the time for that in the neuropsychiatric ward, and therefore it seems to me that it is much better for

the psychiatrist and for the medical officer who must do psychiatry — (and a great many medical officers will have to do psychiatry with little preparation) — that they view the neuropsychiatric casualties from the standpoint of the outstanding symptomatic feature.

My experience with neuropsychiatric casualties leads me to say that they fall into three great groups, from the standpoint of what may be seen clinically, the important segment of the disability. These three groups are as follows:

First, there is a *physical group* in which the outstanding symptoms, though functionally determined, are nevertheless completely physical. They are the soldiers who have convulsions and coma, paralysis, deafness, blindness, headache, tics and anesthesia, and so forth. Those are the symptoms of the disability.

In the second group, which is an *emotional group*, the thing you see there which strikes you is the emotional segment of the clinical picture — depression, often suicidal, elation, resentment, tremendous fear, irritability, suspicion, excitement, anger, rage and so forth.

The third group is a group in which you see the soldier because he has been guilty of military misbehavior. That is the important aspect in that group. This group includes men who have been suspected of Fifth Column activities or of spying or of inciting to insubordination or of striking an officer, drunkenness, negligence, grousing, quarreling and petty stealing.

While it would be very nice to think in terms of exact diagnosis and classification, one cannot do that in modern warfare. There is'nt the time. However, one gets pretty far along if one has these three groups in mind.

Nevertheless, it is necessary for the medical officer to be able to answer a few basic questions before he can do anything for the neuropsychiatrically disabled soldier. The examination, sketchy as it must be, is compelled to be sufficiently inclusive so that in the majority of cases, these questions

can be answered. These questions are: Is this disability organic or is it functional? If it is organic, is the seat of the disability outside of the central nervous system? It might be a uremic convulsion or acute sinusitis. If it is functional, is it a psychosis? Is it mental disease or is it a psychoneurosis? And if it is a psychoneurosis, is it the usual and common psychoneurosis of war, which is conversion hysteria, or is it one of the less frequent neuroses like a neurasthenia or an anxiety state, or an obsessive compulsive reaction? Can the disabling symptom be speedily removed? This is of the utmost importance, as I will presently develop for you. Finally, it is malingering?

It is useless for the medical officer doing psychiatry and for the psychiatrist in the army to expect to have any elaborate history. There won't be any. The best information will come from the top sergeant or one of the other sergeants, or from one of the buddies of the disabled man. Some kind of a neurological examination should be made.

In the first World War, we reduced the neurological examination to a few things, which could be done in less than two minutes: Temperature, the pulse, the pupillary responses, particularly to light, the tendon reflexes, chiefly the knee jerks, the abdominal reflexes, the Romberg test, the posture and the gait. That was about all for which there was time. Yet it was amazing how many neurological cases were picked up in this very brief examination—quite a sprinkling of paretics and an amazing number of cases of disseminated sclerosis, which is not an uncommon disease as the neurological textbooks say, but quite common, particularly at the age epoch which is the military age.

The psychiatrist and the military officer doing psychiatry with the armed services, do get a few breaks. They do not have to worry about the psychosis of senility; at least, they are not going to be allowed to examine anyone who might have a psychosis of senility, but they do not have to bother about that. Certain things are excluded by military age limits.

Perhaps the chief information obtained from the examination is derived from what the psychiatrist can see when he looks at the disabled man. This is true whether the disability occurs at an encampment or whether it occurs within a half-hour of its development in the theatre of battle. There is much more to be learned by looking than there is by asking a great many questions, such as, "do you feel depressed, and are you unhappy?" Everybody in war is unhappy so that question isn't of very much importance anyhow. The things that can be seen by looking at the man, by observing his general appearance, the state of his clothing, his facial expression, which is of the utmost importance, his general motor attitude, whether it is one of cringing fear or whether it is one of aggressive anger and determination; whether it is cataleptic; whether he is in stupor; whether he is negativistic and so forth and so on.

However the examination is made, and it must be made quite rapidly, the important thing to determine is the emotional state of the individual. An estimate of the emotional state and of consciousness is a necessary condition of diagnosis and treatment, irrespective of whether you are dealing with psychosis or psychoneurosis or malingering.

I referred at the beginning of these few remarks to the fact that the incidence of neuropsychiatric disabilities is unquestionably the most serious and important disability consideration in the war. I said that I could not give figures, but figures that may be given from the last war, from all the armies—and they will not be less in this war—showed that one-seventh of all disabilities were neuropsychiatric. If one excludes wounds, then one-third of all disabilities are neuropsychiatric, so even considering surgery and the many other important medical aspects of war, the conclusion that the neuropsychiatric problem is the most important one is inescapable.

There is something over and beyond that cannot be figured in terms of statistics, and that is, the effect of neuropsychiatric

disabilities upon morale. Morale is a strange and odd thing. It usually does not work the way one expects it to work. I am tempted to diverge for a minute, (thinking of some of the previous speaker's remarks about morale, and the excellent morale of the civil population of the much-bombed cities of England) to tell you a story which perhaps illustrates that morale works in strange ways. It was told to me by Wing Commander Gillespie of the RAF.

An old lady who lived all her life in the slums of London in a neighborhood that was very much bombed. She took the bombing in her stride, without the least concern or difficulty. Finally the particular row of tenements, in which she lived, was reduced to such a rubble that the authorities insisted she be evacuated and moved to the country. She protested very much, but finally, by a mixture of military order and persuasion, she was induced to go to the country with other evacuees.

As they were in the train, going on to their destination and passing through the countryside, she suddenly passed out. The reason she fainted was because she saw a cow which had come somewhere near the tracks of the train. After she was brought to with stimulants, they said to her, "Why, you stood the terrible bombing in London, you never gave a whimper but went about your daily work. How do you explain it?" She said, "Oh, that's the ordinary, everyday thing! Bombing, that's nothing, that's a natural thing. But such a horrible, supernatural, strange animal as I saw here in the country—that's more than it is reasonable to ask any person to stand!" Left untouched by the bombing of London, her morale was upset by a cow.

Well, I said there was something that could not be expressed in figures of neuropsychiatric disabilities but which was extremely important, namely, their effect on morale. I think perhaps the Colonel will agree with me that the morale effect of a wound, especially a wound on the field of battle, even though it be a mortal wound, is good. It produces or activates emotions which are necessary so that the soldier will

kill with a will. This is necessary in war. The soldier becomes angered and enraged because one of his friends has been wounded. As the result of what he has seen, he becomes a better fighting machine.

On the other hand, if, on the field of battle, as frequently happens, a soldier falls to the ground and twitches in an hysterical fit or has some other major neuropsychiatric manifestation, it is destructive and disruptive of morale. There is something strange and mysterious about fits without wounds. There hasn't been any wound which put the man down. It is odd and queer and disturbing. It even gives rise to thoughts that there is some mysterious weapon which can accomplish these almost supernatural effects. In any event, all in all, it is extraordinarily bad for morale, and therefore you may say that in addition to the incidence, which is great enough as it is, there is to be added the very strong disruptive impact of neuropsychiatric disabilities upon the morale of the troops witnessing them.

I can only touch high points here and there. In the psychiatry of civil life, disturbances of consciousness and disorientation, very often have rather serious import. In general, in war, in military neuropsychiatry, their prognostic significance is good. In the small World War in which I participated, we were quite puzzled for a while by soldiers who were brought in with an acute psychosis, showing a complete severing of consciousness, tremendous hallucinosis and motor activity. They looked to us like situations we saw in civil life—acute schizophrenic panics and so forth—and we thought the import was ominous.

We found that in a large percentage of those reactions which deserve to be called acute war psychoses, a number of days of rest and good hot food was sufficient to produce a complete recovery. This was a rather astounding difference between military and civilian psychiatry.

There was a large element of exhaustion in acute war psychoses, and they responded exceedingly well to simple

treatment of hot food, increase of fluid intake, rest and then a very elementary sort of explanation.

What about the malingerer? I think a rather erroneous idea exists as to what constitutes a malingerer. I have a theory that sometime during his military life, every man and every officer is potentially a malingerer.

The previous speaker referred to something that I think is of the utmost importance in this particular problem of malingering. A whining letter from home often makes a potential malingerer, which we all are at some time, into an active one. A letter from home, in the case of an unmarried man, for instance, from one of his so-called friends, saying, "I saw your girl out last Saturday night in the park with a couple of fellows," is enough to turn the tide very often, especially if the boy is already homesick and worried; or from a wife, saying, "I don't see how I can possibly get along with the allotment; the children are sick, so am I but I hope you are well" or a kind of "the hell with it anyhow" letter is enough to make the potential quality of malingering (which is in each one of us) into an active thing.

This kind of malingerer, if handled reasonably skillfully (and it requires no particular technique to do that and I think the good commonsense medical officer does it as well or perhaps better than the psychiatrist) constitutes no real problem. A little talk with those boys, divested of any thought that "maybe I'm going to have you court-martialed," just a sensible talk, is quite enough to turn that kind of malingerer into an extremely good soldier. Very often, the experience after such a talk is that the boy says, "Well, I guess that's O. K. I see that. Don't mention it to anybody. I'll go ahead and do a good job."

But if it were possible to look back at the records of that man in civil life, you would find he was a real fellow in civil life. The kind of malingerer who is going to give trouble, and whom the army doesn't want and is glad to get rid of, is the

malingerer whose record in civil life is also one of malingering against anything—against social conditions, occupational conditions and so forth. Such a man may have a small prison record. He is a rebel against society. That kind of a malingerer is not bothered by disturbing letters from home. He is the man who, from the moment he is in the army, is making a deliberate, steady, practical, crafty attempt to pretend; to feign illness and to act in such a way that he will get his military discharge. That kind of malingering is bad. There isn't too much of it, and it is absolutely useless to try to treat such a malingerer.

You recall the three great groups in which I think neuropsychiatric disabilities fall; one, presenting physical signs of importance, like paralyses; the second, the emotional group; and the third, those who are up for military misbehavior.

In the last group, the military misbehavior, there is often evidence of an incipient psychosis, or psychoneurosis or even of organic neurological disease. I remember a court martial in France after the last war in which a boy was tried because he had been one of a party of five sent through No Man's Land to get information. He was in the middle of the human chain. The custom was to hold hands and crawl across on their bellies. About half-way over, he gave a shriek, which, of course, drew the fire of the enemy, and three of the men, not including the boy, were killed.

He was tried for cowardice which, of course, in action is punishable by death. It was a rather long session, and some evidence from his civil life was brought in. The man should never have been admitted into the army. He had epilepsy. He had had an epileptic attack under emotionally trying circumstances, and epileptics often do have their attacks under emotional strain. The boy was acquitted.

Therefore, military misbehavior may have a very deep significance. The common war neurosis in the last World War, and I think to a considerable degree it will be so in this war, is what may be called conversion hysteria — the shell shock

of the newspapers. It was typically the neurosis of the man in the ranks.

We felt, after a good deal of experience, and particularly with the experience permitting us to see these so-called shell shocked soldiers, sometimes within less than fifteen minutes of the development of the shell shock, that we had some idea of the mechanism underlying conversion hysteria.

The previous speaker referred to tirage, I think. We operated at what was called a tirage. The casualties passed through in great numbers with the orthopedist, the shock man (whose resources were much less adequate than they are now) and the psychiatrist. Our chief work was not to treat but to attempt to sort out those patients who, by the nature of their disability, would hold up military operations too long. They were sent back to Paris or some other hospital center.

Those who had a reasonable chance of recovering within the period demanded by the military action — which is of the utmost importance and was fixed by headquarters — remained within a few miles of the front and were treated there. Amazingly, with few treatment facilities and crude methods, with nothing much more than suggestion therapy, they recovered in far greater numbers than in conversion hysteria in civil life. The answer, of course, was that they were treated while, as we called it, the neuroses was still "warm" in their making.

Conversion hysteria is always the result of emotional conflict, conflict between two things that are irreconcilable, so that a pathological solution of hysteria may eventuate. In war one element of the conflict is the demand of the instinct of self-preservation which is inherent not only in man but in all biological structure. War or patriotism does not magically remove such an inherent and innate instinct.

If you are crossing Seventh Avenue in a hurry and perhaps thinking deeply of some life insurance problem, and taking chances on the traffic, suddenly you jump to one side. The reason you jump is because you were in dangerous prox-

imity to an automobile. Its warning horn sounded and immediately your instinct of self-preservation became vigilant, and removed you from the field of danger. You jumped out of the path of danger. So, in every man in war, who is in the sector of combat, man or officer, his instinct of self-preservation is very much on duty.

Opposed to that, and at first glance, irreconcilable with it, are a group of things that might be grouped together under "soldierly ideals", ideals of soldierly duty, patriotism, honor, training, disciplinary reactions, and so forth. I think it is fair to state that in effect, a man cannot be a good soldier without endangering his life, so that in everyone who is in the theatre of combat, there is this constant, underlying, unconscious conflict going on. I don't mean that men and officers, as they saw each other at the beginning of a day, said, "How's the instinct of self-preservation today? Do you think it's going to give you much trouble? How are your soldierly ideals functioning?" But, nevertheless, there was an underlying and constant conflict, and under certain circumstances which provided the precipitating reason for the conversion hysteria and which very often, in the first World War, was being bowled over by the concussion of a shell without injury, but might also be great fatigue, deprivation of food and water—the conflict came to its pathological compromise, and the emotional friction was converted into certain disabling physical signs and symptoms, which removed the soldier from the sector of danger.

It was interesting to note how closely the conversion symptoms were in line with the situation in which the soldier had been placed. A very common type of conversion hysteria was the kind in which the soldier was brought into the ambulance dressing station or tirage, totally blind and amnesic. "Well, the last thing I remember is being told to charge across that wheat field. I think I charged, but that is the last thing I remember. The next thing I remember is being picked up by the stretcher bearers."

You see how beautifully his conversion symptoms protected him from a situation which his personality could no longer face. Blindness, probably, and sometimes we verified it, because he had seen the head of one of his friends blown off by a shell, and amnesia because his psyche could no longer bear to remember what he had seen. And so did we have hysterical deafness in those who had been left exposed on the field of battle listening to the cries and groans of the wounded. And in a very small and interesting group did we have the hysterical loss of the sense of smell in men detailed to burial parties to bury the bodies of men and horses. Because of the activity of the military operation, these bodies had been lying above ground for a long period of time, so it is only fair to describe their condition by a robust Anglo-Saxon word—they were stinking. So some of the soldiers detailed on burial parties were almost magically protected from the horrible stench by the development of a hysterical loss of the sense of smell.

In many soldiers, tics of the belly muscles, which, on first glance, looked like some obscure neurological disease, had such a simple explanation as this: They had run their bayonets through the bellies of enemy soldiers, and the final neuropsychiatric result was that they developed these amazing tics of the whole wall of the belly.

This type of conversion hysteria occured usually in men in the ranks, the soldier who was average intellectually, while the other neuroses, neurasthenia, anxiety and compulsive-obsessive reactions occured in the superior soldier and in the officer.

The symptoms of the type of hysteria I have described appeared much more abruptly than in the other neuroses, and the symptoms were usually total symptoms — blindness instead of spots before the eyes, such as you see in neurasthenia. Often, these conversion symptoms were removed by the simplest kind of suggestion, if the case was seen soon enough. I think that is going to be true this time. I think having

Blitz-krieg warfare instead of trench warfare will make no difference excepting that the time element will be all the more important. There was a direct ratio between the chronicity of the case and the time which had elapsed between the occurrence of the conversion symptoms, and the attention of a medical officer.

These are highly important considerations, notably during combat, when time limits for restoration to duty are very often only a matter of a few days and are issued by headquarters; in a certain engagement, you might have three days to try to get the neuropsychiatric cases well. If your judgment was that they could not get well in three days, then back to Paris for that group. Otherwise, the military machine would be impeded.

What is said of first aid in the combat zone applies with equal force to soldiers in encampments and in quiet sectors. In the zone of active combat, the neurosis will be much more severe. There will be fewer and poorer treatment facilities, yet the results, if the disabilities are seen soon, will be better than in civil life, and the restorations under the one important condition, of quickness in seeing the patient, will reach 70 per cent.

May one be at all hopeful about the prevention of neuropsychiatric disabilities? Certainly not, from the standpoint of complete prevention. That would be as inaccessible an ideal as it would be to say that we will prevent head wounds in war. But from the standpoint of reducing the incidence of neuropsychiatric disabilities now alarmingly high, there is a great deal to be hoped for. The best prevention is at the source, and by that, I mean that unless reasonable care is exercised in not admitting or inducting into the army men who obviously are poor neuropsychiatric goods, why, then the incidence must be very high. Possibly you have been told that psychiatrists ask for impossible considerations in the induction examinations. I may tell you that they don't. They want about ten or twelve minutes to determine whether

a man is likely to break neuropsychiatrically or not. That does not seem too much, and two minutes is too little, and the more two-minute examinations there are, the greater is going to be the incidence of men who are neuropsychiatrically too fragile to stand the situation of modern war. They will break.

I should say, in the last analysis, what does it profit a nation if it has a huge army if that army is not a fit army? I have no hesitancy in saying that the chief way to see that that is a reasonably fit army is not to admit into that army weak, breakable human material, and that is particularly true of neuropsychiatric material.

And then, when you have a reasonably good army, you can still do something in the way of prevention of too many neuropsychiatric disabilities. That is something we are working on—at least part of it may be told. I think it is quite possible and feasible to condition men against at least the surprise element of the impact of war. After all, what might be called the stimuli which are operating and producing neuropsychiatric disabilities are not many. They are: first, noise, detonation, horrible, strange and amazing noises of all kinds, not only in volume of sound but also in the nature of sound, queer shrieking noises of all kinds.

Then there is illumination, which is certainly an agent in the production of neuropsychiatric disabilities—sudden, frightening illumination.

And then there was, and I am not enough of an optimist to say that there won't be this time, even though there hasn't been yet, poison gas, which is an element in the neuropsychiatry of the war.

I think it is conceivable to condition men by exposing them in miniature to these major stimuli of the war, so that at least they have some kind of vaccination against it. Then their first contact with the frightening hazards of war will be less likely to smash their nervous systems. If we have in mind not only an army of volume but also an army of fitness,

plus a reasonable amount of conditioning to the neuropsychiatric hazards of war, we will easily defeat the enemy.

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PRESIDENT WENSTRAND—Gentlemen, Dr. Strecker has told us a great many interesting things. I do not believe that we should bother him with any questions, unless there is something very important that one of you has to have settled.

DR. HARRIS M. GRAY—Mr. Chairman, if I might interrupt, if it would be of interest to the meeting, I would be very glad to say a few words about the handling of the neurosis in England, because of rather close personal association with the consultant of the Canadian Army, and also a close personal association with one of those operating under the EMS, handling these neuroses. If it would be of interest, I would be glad to say a few words about it.

I am sorry to interrupt, but I was rather stimulated by Colonel Gordon's speech about England, and it brought it all back to me.

While there, I had a chance to watch how they actually were handling these neurosis cases, and the type of neurosis that they were running into. Contrary to what was expected, we did not have very much neurosis developing from these blasts and the Blitzkrieg and the bombing and so on, and most of the question of neurosis was very easily settled and quickly disposed of, and returned back to Canada, as far as the Canadian soldier was concerned.

These men were found to be those who had had limited ability and experience in life, and had never really been able to cope with things in the past, or had lived such a limited sort of existence that when they were intended to broaden out, they had to be returned. Others, also with poor background, particularly poor attempts at schooling, were the ones that broke down under the emotional stress of family troubles at home and these depressing letters.

These people were returned immediately to Canada as unfit for further service. One reason for that is that the type of warfare going on in Great Britain at the present time and

since the beginning of the war was one in which the individual man had to act as an individual. They had to depend on him. In many places, the individual was armed to the teeth and left alone for twenty-four hours to hold a vital spot, and they could not have a man who might break down. Therefore, cases that perhaps might have otherwise been allowed to carry on in some minor capacity were disposed of at once.

With regard to the Emergency Medical Service, the British Medical Association did rather a remarkable thing in this organization of the doctors. They had them listed according to their ability. It was my privilege, while doing some extra study in London, to stay for five weeks with one of these first-string neuropsychiatrists, and I was able to see the operations of the system in his particular case.

He was a man who, after some twelve years of post-graduate work, had gotten pretty well up towards the top. He was fully employed from nine o'clock in the morning until six o'clock at night with hourly appointments, at a guinea a visit, and he was listed with the Emergency Medical Service. He got his call eventually when the need arose for further men, and was given two weeks in which to wind up his affairs, close out his place in London, his splendid offices and everything else, and then moved at the direction of the Ministry to the spot where he was most valuable.

There, he carried on this work, and most of the patients really were ones whom the medical officers had been unable to cope with, and for the reason of the importance of the individual in this present warfare, had been evacuated to the special centers. He was still a civilian. He received the pay of a Major, and without any extras, which meant that he had to live very simply. As a matter of fact, he moved from his fine residence on Harley Street to what was called a miner's cottage in Sheffield. The contrast was extraordinary. But he was very happy in his work and working very hard.

As an example of how that system worked and how efficient it was, they took alternating afternoons with what was

known as an out-patient clinic. To this clinic were sent the soldiers from that military area for disposal of these neuroses, and his average number of cases handled in one afternoon in the clinic would be about fifteen.

I was struck by the pithiness of his summaries of these cases, which I was able to read, as each afternoon clinic was completed. They were completely disposed of. In most cases, they had to be returned to civil life and fitted in at their former occupations. But that was the final opinion, the Army acted on it, and they went on about their jobs.

I thought this might be of some interest to the members, and so I wished to present it to you.

PRESIDENT WENSTRAND—This brings to a close our meeting. We have many things to think about, and I have been delighted with the way that things have gone. I want to thank you all again for having given me this opportunity and this privilege.

Dr. William Bolt, will you step forward, please? Dr. Bolt, it gives me great pleasure to hand over to you the gavel.

PRESIDENT-ELECT BOLT—Thank you, Dr. Wenstrand!

Gentlemen, I thank you for the honor you have conferred upon me by electing me your President. I can assure you that I will do all in my power to be worthy of your confidence.

It is getting late now and we don't want to delay any longer. Is there any other business to come before the meeting?

DR. ROWLEY—At this point in the proceedings of the annual session of this Association, it has been my privilege on other occasions, as it is again now, to act as your spokesman in expressing to our retiring President the feeling that I know is in the hearts and minds of each one of you, that we owe him a great debt of gratitude for preparing and carrying through for our benefit such a successful meeting as this has been.

In ordinary times, I know that it is no easy task to arrange the program for these meetings and to obtain the speakers, and at this time, which is not a normal time, it must have been a task of even greater magnitude. But he has risen to the oc-

casion most splendidly and has given us one of the best meetings that, I think you will agree with me, we have ever had.

While no formal vote is necessary to express the thought, I am sure you will join with me in the thought that our gratitude to Dr. Wenstrand, our retiring President, should be expressed in fullest measure.

PRESIDENT-ELECT BOLT—Gentlemen, I am sure it is not necessary to put that to a formal vote. You have indicated your thorough agreement with Dr. Rowley's sentiments.

Before we adjourn, I want to mention just one thing. Dr. Dewis has told me that there are several members here who have not yet registered. If there are any in the room who have not registered, will they please do so at the desk before they go out, so we may have a complete record of those who were present at the meeting.

If there is no other business to come before the meeting, a motion to adjourn is in order.

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Upon motion regularly made and seconded, the meeting was adjourned.

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The following members, delegates and guests were present at some time during the sessions: Doctors W. J. Allison, K. W. Anderson, T. D. Archibald, E. M. Armstrong, T. M. Armstrong, W. B. Aten, J. A. Avrack, H. A. Bancel, G. H. Barber, N. J. Barker, W. B. Bartlett, E. W. Beckwith, J. E. Bee, M. B. Bender, E. B. Bigelow, C. C. Birchard, William Bolt, E. C. Bonnett, J. T. Borman, W. M. Bradshaw, K. F. Brandon, A. W. Bromer, C. T. Brown, H. B. Brown, B. F. Byrd, E. J. Campbell, M. F. Campbell, F. H. Carber, P. E. Carlisle, V. S. Caviness, J. P. Chapman, E. D. Chesebro, B. R. Comeau, P. M. Cort, D. B. Cragin, H. W. Crawford, J. H. Crawford, R. M. Daley, A. H. Davison, H. D. Delamere, P. G. Denker, E. J. Dewees, E. G. Dewis, T. H. Dickson, E.

S. Dillon, H. W. Dingman, W. W. Dinsmore, J. L. Evans, W. G. Exton, E. W. Fahey, J. G. Falconer, H. H. Fellows, W. E. Ferguson, J. H. Font, R. A. Fraser, H. M. Frost, F. I. Ganot, D. S. Garner, J. T. Geiger, W. M. Genthner, J. M. Gilchrist, R. A. Goodell, J. K. Gordon, H. M. Gray, George Greenway, F. L. Grosvenor, R. S. Gubner, Llewellyn Hall, Frank Harnden, L. E. Hathaway, W. C. Hausheer, H. M. Hawkins, J. K. P. Hawks, W. D. Heaton, D. W. Hoare, C. O. Hollinger, Gilbert Horrax, J. L. Humphreys, J. H. Humphries, J. E. Hunsinger, S. W. Hurdle, J. R. B. Hutchinson, C. B. Irwin, W. A. Jaquith, A. O. Jimenis, A. E. Johann, W. M. Jones, G. E. Kanouse, E. F. Kerby, E. A. Keenleyside, H. B. Kidd, C. E. Kiessling, D. G. Kilgore, C. T. Kirchmaier, W. P. Lamb, P. H. Langner, I. C. Lawler, J. M. Livingston, H. C. McAlister, C. B. McCulloch, A. J. McGanity, Robert McGrath, George McLean, L. L. McLellan, R. E. McLochlin, F. W. McSorley, H. R. Magee, H. L. Mann, R. W. Mann, W. L. Mann, J. F. Moore, Jr., A. E. Murphy, C. F. Nichols, Eduard Novak, Jan Nyboer, A. J. Oberlander, M. J. O'Brien, Herbert Old, W. O. Pauli, C. B. Piper, J. J. Post, O. S. Randall, E. G. Reinartz, P. V. Reinartz, W. A. Reiter, D. C. Roberts, A. J. Robinson, H. B. Rollins, T. F. Ross, R. L. Rowley, B. T. D. Schwarz, E. W. Scott, J. T. Sheridan, A. L. Sherrill, D. M. Shewbrooks, R. R. Simmons, J. L. Siner, Morton Snow, J. B. Steele, I. R. Stidger, E. A. Strecker, S. J. Streight, N. A. Sullo, E. V. Sweet, L. G. Sykes, B. C. Syverson, H. M. S. Tait, G. F. Tegtmeyer, W. E. Thornton, G. D. Townshend, I. G. Towson, Joseph Travenick, Jr., H. B. Turner, H. E. Ungerleider, R. C. Voss, W. R. Ward, D. E. W. Wenstrand, S. S. Werth, J. F. Whinery, C. F. S. Whitney, H. E. Wiley, R. L. Willis, E. B. Wilson, F. N. Wilson, M. C. Wilson, G. E. Woodford, L. S. Ylvisaker, D. E. Yochem, E. T. Yorke and Messrs. L. F. Baker, R. E. Fletcher, Arthur Hunter, Edward King, E. A. Lew, Montgomery Schuyler, P. G. Shepherd, O. G. Sherman, P. V. Wells and A. C. Webster.

Total attendance at all sessions, 179.

MEMORIALS

DR. WILTON FISHER BLACKFORD

1873–1942

Dr. Wilton Fisher Blackford, aged 69 years, was born in Olney, Illinois, and died April 2, 1942. His early life was spent in New Washington, Indiana, where he attended school and afterward taught school there for five years.

He attended the Kentucky School of Medicine, from which he received the degree of Doctor of Medicine in 1901. After a five-year period of practice of medicine in Louisville, he joined the staff of the Commonwealth Life Insurance Company as a medical examiner. In 1906 he was appointed Assistant Medical Director and became Medical Director in 1913, which position he held continuously until his death.

He played an important part in attaining the position now occupied by the Commonwealth Life among the life insurance companies of America.

The outstanding underwriting record of his company over the years, stands as an eloquent testimony to Dr. Blackford's unusual capabilities.

He had been a member of the Medical Section of the American Life Convention since 1913 and was elected to the chairmanship of that organization at the last meeting he attended, in June, 1941.

He had been a member of the Association of Life Insurance Medical Directors for many years.

Dr. Blackford's sterling qualities endeared him to all those associated with him. He was a true and loyal friend, unselfishly adding much to the happiness of others.

He will be greatly missed by all who were fortunate enough to know him.

DR. DAVID N. BLAKELY

1867–1942

We regret to announce the death of Dr. David Newton Blakely, Assistant Medical Director of the New England Mutual Life Insurance Company.

His health had been somewhat impaired for several months but he was on full-time duty until within a few days of his death, which occurred on October 15, 1942.

Dr. Blakely was born in Campton, New Hampshire, March 3, 1867, one of the six children of the Rev. Quincy and Gertrude (Sykes) Blakely. He spent three years at St. Johnsbury Academy (Vermont) graduating in 1885. He then entered Dartmouth College and graduated in 1889. In October, 1889, he went to Aintab, Asiatic Turkey, where he spent four years associated with the American Board of Foreign Missions and the Central Turkey College. His work had to do with the accounts and business details of the organizations. On his return to America he entered Dartmouth Medical College from which he graduated in 1896. After graduation he interned at the Boston City Hospital and followed this with two more years as Assistant Resident and Executive Assistant.

He entered private practice in Boston in 1900. For several years he was on the staff of the Boston Dispensary and the Massachusetts General Hospital and the Infants' Hospital and was school physician under the Board of Health. He also taught histology at Harvard Medical School and was instructor in physical diagnosis at Tufts Medical School.

From 1900 to 1917 he was examiner for the Metropolitan Life Insurance Company. In 1907 he was appointed Home Office Examiner of the New England Mutual Life Insurance Company, in 1910 Assistant Medical Director (half-time basis) and in 1917 full time. He was elected a member of the Association of Life Insurance Medical Directors in 1912.

His "Urinalysis: A Review of Ten Years' Experience of the New England Mutual," page 419 in the 26th Annual Proceedings of the Association of Life Insurance Medical Directors, 1915–16, was a notable contribution. He also made another contribution, "Discussion of Diabetes as a Life Insurance Selection Problem." Proceedings: Vol. XVIII—page 283.

He joined the Massachusetts Medical Society and the

American Medical Association in 1898. He became a member of the Clinical Club in 1905 and served as secretary from 1907 to 1924. In 1924 he was elected president of the Norfolk District Medical Society and was chairman of the Committee on Membership and Finance of the Massachusetts Medical Society from 1922 to 1938. He was secretary and treasurer of the Dartmouth College Class of 1889 from 1914 until his death.

He married Miss Amy Burrage in 1907 and had two children, Elizabeth and John, all of whom survive him.

He was a member of the Appalachian Mountain Club, and a Mason, and was active in the affairs of the Unitarian Club and the Unitarian Laymen's League.

Dr. Blakely was particularly skillful in writing letters to policyholders who had been given health examinations. These letters, characterized by felicitous turns of expression and appropriate examples giving point and force to his advice, seemed to carry individual personal messages.

The problems connected with the selection and appointment of medical examiners and acting as arbiter between examiners and agents which came in his province were handled with tact, understanding, fairness and good judgment.

Dr. Blakely was a kind, sincere, faithful and loyal friend. His manners were gracious, warm and genial. He was kind, generous and considerate in his judgments. His was a cheerful spirit; his mood was one of uniform, quiet, good humor. He was deeply religious. His ideals were high, his character sterling. He was modest and unassuming. He was highly esteemed and will be greatly missed.

DR. CHARLES E. ILIFF

1874–1942

Dr. Charles E. Iliff died after a short illness on July 11 at Cincinnati, Ohio.

Dr. Iliff was born in Cincinnati on July 9, 1874. He received his preliminary education at the public schools in that

City and his Medical Degree from the Miami Medical College in the year 1900. After serving his internship at the Cincinnati General Hospital, he engaged in the practice of his profession in his native city.

He became associated with The Western and Southern Life Insurance Company as a medical examiner early in his career. He was appointed Associate Medical Director of the Company in 1920 and Medical Director in 1930, serving in that capacity until the time of his death. He was intensely interested in the problems and welfare of the field force and his Home Office associates. His kindly manner and cheerful greetings will be greatly missed by them.

Dr. Iliff was a member of the visiting staff of the Jewish Hospital and of the consulting staff of Good Samaritan Hospital. He was also a member of the Ohio Medical Association, Cincinnati Academy of Medicine, American Medical Association, American Public Health Association, Association of Life Insurance Medical Directors, Medical Section, American Life Convention and Past President of the Cincinnati Obstetrical Society.

He was a Syrian Shriner, a member of the Scottish Rite and Vattier Lodge of the Masonic Order.

Dr. Iliff is survived by his widow, Mrs. Frances Jessie Haag Iliff; a son, Dr. Charles E. Iliff, Jr., associated with the Johns Hopkins Hospital in Baltimore; three sisters, Mrs. William McMiller, Akron, and Mrs. Gertrude Hogan and Mrs. Gilbert Steinau, Cincinnati, and two grandchildren.

DR. FRANK W. KENNEY

1860–1941

It is with deep regret that the death is recorded of Dr. Frank Woodbury Kenney, Medical Director of The Capitol Life Insurance Company, Denver, Colorado, which occurred at his home on December 19, 1941, following a long illness.

Dr. Kenney was born in Athol, Massachusetts, on October 25, 1860, receiving his early education in the schools of that

place, as well as in Lynn and Salem. It may be said of him that his education was obtained in the difficult way that befell young men of that day who had scant means and yet who determined to follow a profession. His medical training was completed at the University of the City of New York, and for a time he practiced in the East. Ill health demanded a change of climate, and the young physician turned his face westward, locating in Denver. He found it a city well suited to his tastes. Here he regained his health, and was soon prominently identified with the leading medical men of the City and State. For thirty-nine years he enjoyed a splendid practice, and commanded the respect and affection of his fellow doctors, taking an active part in all affairs tending to the advancement of the profession. To him may be credited the building up of the outstanding medical library which is the property of the Denver County Medical Society, and to which he gave much time and intelligent planning, not alone to its compilation, but to its housing as well.

With his New England heritage of stern virtues, conservative judgment and cultural endowment, Dr. Kenney happily amalgamated the friendly hospitality and liberal outlook of the West. A gentleman of the old school, punctilious in his habits, wholly devoted to his duties, whatever they might be, of keen intellect, and yet extremely modest, he possessed that rare charm which endeared him to his friends. Remarkably youthful in appearance, and even more so in his outlook on life, he never found time in his busy career to grow old. He was keenly interested in those with whom he was associated, as well as the affairs of the day. He had travelled extensively, and was a delightful conversationalist.

Although his last illness confined him to his bed less than six months, he had known for several years that he was afflicted with a malady for which there could be but temporary relief. He met it courageously, welcoming his friends to his bedside, and maintaining his cheerfulness and his unfailing interest in those friends.

Dr. Kenney's passing has brought sadness to many hearts—

those of his beloved friends, his patients, and to those who have been associated with him during the time he has been Medical Director of The Capitol Life. He is survived by his wife, who resides in Denver.

DR. WALTER T. McNAUGHTON

1880–1942

Dr. Walter T. McNaughton, Medical Director of The Old Line Life Insurance Company, died of a heart attack in his office on July 7, 1942.

Although born in Ottawa, Ontario, June 15, 1880, Dr. McNaughton referred to himself as a "native" Milwaukeean. His father, one of the early Wisconsin contractors, was in Ottawa on a construction project at the time of his son's birth, but the family returned to its Milwaukee home six weeks later.

Educated in Milwaukee schools, he in 1904 received his medical degree from the old Wisconsin College of Physicians and Surgeons. After graduation he acted as interne in an Oshkosh hospital and then returned to his home town to practice. During the first World War he served as a Lieutenant in the United States Army Medical Corps. In 1928 he became Assistant Medical Director of the Old Line Life and two years later was appointed Medical Director.

Dr. McNaughton was active in the Masonic Order and was a member of the Cudworth Post, American Legion. He was a member of the Milwaukee Academy of Medicine and other medical societies: county, state and national. He belonged to Phi Rho Sigma medical fraternity.

Dr. McNaughton was always friendly, accommodating, and a good companion. He was held in high esteem both among his patients and his business associates. His profession held his interest above all else.

He is survived by his wife, a brother and a sister.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Wilton F. Blackford, M. D.	Louisville, Ky.
David N. Blakely, M. D.	Boston, Mass.
Robert J. Blanchard, M. D.	Winnipeg, Man.
Frederick G. Brathwaite, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York, N. Y.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Henry Colt, M. D.	Pittsfield, Mass.
Henry W. Cook, M. D.	Minneapolis, Minn.
Thomas C. Craig, M. D.	New York, N. Y.
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
John W. Fisher, M. D.	Milwaukee, Wis.

DECEASED MEMBERS

Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Homer Gage, M. D.	Worcester, Mass.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Frederick L. Grasett, M. D.	Toronto, Can.
Landon Carter Gray, M. D.	New York, N. Y.
Frederick W. Hagney, M. D.	Newark, N. J.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Joseph B. Hall, M. D.	Hartford, Conn.
Edward H. Hamill, M. D.	Newark, N. J.
William W. Hitchcock, M. D.	Los Angeles, Calif.
Angier B. Hobbs, M. D.	New York, N. Y.
Donald C. Hoffman, M. D.	New York, N. Y.
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Ross Huston, M. D.	Des Moines, Iowa
Henry H. Hutchison, M. D.	Toronto, Can.
Lefferts Hutton, M. D.	New York, N. Y.
Charles E. Iliff, M. D.	Cincinnati, Ohio
Phineas H. Ingalls, M. D.	Hartford, Conn.
Arthur Jukes Johnson, M. D.	Toronto, Ont.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
Frank W. Kenney, M. D.	Denver, Colo.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.

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Henry P. Lyster, M. D.	Detroit, Mich.
Milton T. McCarty, M. D.	Frankfort, Ind.
Charles N. McCloud, M. D.	St. Paul, Minn.
Francis A. McGreen, M. D.	New York, N. Y.
Lewis McKnight, M. D.	Milwaukee, Wis.
Thomas H. McMahon, M. D.	Toronto, Ont.
Walter T. McNaughton, M. D.	Milwaukee, Wis.
Elias J. Marsh, M. D.	Paterson, N. J.
Henry A. Martelle, M. D.	Hartford, Conn.
Allison Maxwell, M. D.	Indianapolis, Ind.
Archibald Mercer, M. D.	Newark, N. J.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William D. Morgan, M. D.	Hartford, Conn.
John P. Munn, M. D.	New York, N. Y.
William Natress, M. D.	Toronto, Ont.
Charles T. Necker, M. D.	Waterloo, Can.
Edwin M. Northcott, M. D.	Portland, Me.
Brace W. Paddock, M. D.	Pittsfield, Mass.
Frank K. Paddock, M. D.	Pittsfield, Mass.
William A. Peterson, M. D.	Chicago, Ill.
Joseph E. Pollard, M. D.	Newark, N. J.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James T. Priestly, M. D.	Des Moines, Iowa
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Oliver P. Rex, M. D.	Philadelphia, Pa.
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Oscar H. Rogers, M. D.	New York, N. Y.
Edward K. Root, M. D.	Hartford, Conn.
James F. W. Ross, M. D.	Toronto, Ont.
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Gurdon W. Russell, M. D.	Hartford, Conn.
George R. Shepherd, M. D.	Hartford, Conn.
Donald W. Skeel, M. D.	Los Angeles, Calif.
Dewitt Smith, M. D.	Dallas, Tex.
George S. Stebbins, M. D.	Springfield, Mass.
George S. Strathy, M. D.	Toronto, Ont.
Melancthon Storrs, M. D.	Hartford, Conn.

DECEASED MEMBERS

Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
Harold F. Taylor, M. D.	Hartford, Conn.
James Thorburn, M. D.	Toronto, Ont.
James D. Thorburn, M. D.	Toronto, Ont.
William Thorndike, M. D.	Milwaukee, Wis.
Paul E. Tiemann, M. D.	New York, N. Y.
Harry Toulmin, M. D.	Philadelphia, Pa.
Frank L. Truitt, M. D.	Indianapolis, Ind.
Henry Tuck, M. D.	New York, N. Y.
John S. Turner, M. D.	Dallas, Tex.
Joseph P. Turner, M. D.	Greensboro, N. C.
S. Oakley Van der Poel, M. D.	New York, N. Y.
Charles A. VanDervoort, M. D.	Philadelphia, Pa.
A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. VanDyck, M. D.	New York, N. Y.
George A. VanWagenen, M. D.	Newark, N. J.
Aaron C. Ward, M. D.	Newark, N. J.
William Perry Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Ont.
William E. H. Wehner, M. D.	Philadelphia, Pa.
Faneuil S. Weisse, M. D.	New York, N. Y.
George R. Welch, M. D.	New York, N. Y.
Ernest A. Wells, M. D.	Hartford, Conn.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
Charles D. Wheeler, M. D.	Worcester, Mass.
A. H. Whitridge, M. D.	Baltimore, Md.
George Wilkins, M. D.	Montreal, Que.
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Gordon Wilson, M. D.	Baltimore, Md.
G. S. Winston, M. D.	New York, N. Y.
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Joseph C. Young, M. D.	Newark, N. J.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

George E. Allen, M. D.	National Life, Montpelier, Vt.
Wilfred J. Allison, M. D.	Southwestern Life, Dallas, Tex.
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Henry H. Amsden, M. D.	United Life & Accident, Concord, N. H.
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Robert B. Andrews, M. D.	Massachusetts Mutual, Springfield, Mass.
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G. Holbrook Barber, M. D.	Manhattan Life, New York City.
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
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James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.

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Maurice B. Bender, M. D.	Guardian Life, New York City.
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
Charles C. Berwick, M. D.	Metropolitan Life, New York City.
Francis P. Bicknell, M. D.	State Mutual Life, Worcester, Mass.
Edward B. Bigelow, M. D.	State Mutual Life, Worcester, Mass.
Cecil C. Birchard, M. D.	Sun Life, Montreal, Canada.
Norman R. Blatherwick, M. D.	Metropolitan Life, New York City.
Harold E. Bogart, M. D.	New York Life, New York City.
William Bolt, M. D.	New York Life, New York City.
Earl C. Bonnett, M. D.	Metropolitan Life, New York City.
J. Thornley Bowman, M. D.	London Life, London, Canada.
William M. Bradshaw, M. D.	Mutual Life, New York City.
Kenneth F. Brandon, M. D.	Aetna Life, Hartford, Conn.
Albert W. Bromer, M. D.	Metropolitan Life, New York City.
J. Lester Brooks, M. D.	Equitable Life, Washington, D. C.
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Frederick R. Brown, M. D.	New England Mutual, Boston, Mass.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Leslie Brown, M. D.	Equitable Life Assurance, New York City.
Bernard C. Bullen, M. D.	Guardian Life, New York City.
Benjamin F. Byrd, M. D.	National Life & Accident, Nashville, Tenn.

Joseph T. Cabaniss, M. D.	Travelers, Hartford, Conn.
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Frank H. Carber, M. D.	Mutual Life, New York City.
Paul E. Carlisle, M. D.	Prudential, Newark, N. J.
Laurence D. Chapin, M. D.	Massachusetts Mutual, Springfield, Mass.
Charles L. Christiernin, M. D.	Metropolitan Life, New York City.
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Augustus D. Cloyd, M. D.	Woodmen of the World, Omaha, Neb.
Edwin A. Colton, M. D.	National Life, Montpelier, Vt.
Parker M. Cort, M. D.	Aetna Life, Hartford, Conn.
Donald B. Cragin, M. D.	Aetna Life, Hartford, Conn.
Hugh W. Crawford, M. D.	Columbian National, Boston, Mass.
Hamilton C. Cruikshank, M. D.	Manufacturers Life, Toronto, Canada.
Robert M. Daley, M. D.	Equitable Life Assurance, New York City.
William L. Davis, M. D.	John Hancock, Boston, Mass.
Harold D. Delamere, M. D.	Crown Life, Toronto, Canada.
Peter G. Denker, M. D.	Equitable Life Assurance, New York City.
Ernest J. Dewees, M. D.	Provident Mutual, Philadelphia, Pa.
Edwin G. Dewis, M. D.	Prudential, Newark, N. J.
Thomas H. Dickson, M. D.	Minnesota Mutual, St. Paul, Minn.
Edward S. Dillon, M. D.	Penn Mutual, Philadelphia, Pa.

Harold W. Dingman, M. D.	Continental Assurance, Chicago, Ill.
William W. Dinsmore, M. D.	Travelers, Hartford, Conn.
Nathaniel P. Doak, M. D.	Great Southern, Houston, Texas
James P. Donelan, M. D.	Guarantee Mutual, Omaha, Neb.
Joseph B. Drummond, M. D.	Union Mutual, Portland, Me.
John T. Eads, M. D.	Penn Mutual, Philadelphia, Pa.
William W. Eakin, M. D.	Standard Life Assurance, Montreal, Canada.
O. M. Eakins, M. D.	Reliance Life, Pittsburgh, Pa.
Joseph E. Engelson, M. D.	Mutual Life, New York City.
John A. Evans, M. D.	Metropolitan Life, New York City.
Francis C. Evers, M. D.	New York Life, New York City.
William G. Exton, M. D.	Prudential, Newark, N. J.
Edward W. Fahey, M. D.	Knights of Columbus, St. Paul, Minn.
James G. Falconer, M. D.	North American Life, Toronto, Canada.
Raymond K. Farnham, M. D.	Metropolitan Life, New York City.
Robert H. Feldt, M. D.	Northwestern Mutual, Milwaukee, Wis.
Haynes H. Fellows, M. D.	Metropolitan Life, New York City.
William E. Ferguson, M. D.	Excelsior Life, Toronto, Canada.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.
Rexford W. Finegan, M. D.	Metropolitan Life, New York City.
Frederick Fink, M. D.	Franklin Life, Springfield, Mass.

Garth E. Fort, M. D.	National Life & Accident, Nashville, Tenn.
John M. Foster, Jr., M. D.	Capitol Life, Denver, Colo.
Robert A. Fraser, M. D.	New York Life, New York City.
Edward M. Freeland, M. D.	New York Life, New York City.
Harold M. Frost, M. D.	New England Mutual, Boston, Mass.
F. Irving Ganot, M. D.	Prudential, Newark, N. J.
Kenneth Gardner, M. D.	Reliance Life, Pittsburgh, Pa.
David S. Garner, M. D.	Shenandoah Life, Roanoke, Va.
John T. Geiger, M. D.	Metropolitan Life, New York City.
William M. Genthner, M. D.	Continental American, Wilmington, Del.
Edson E. Getman, M. D.	New York Life, New York City.
Henry W. Gibbons, M. D.	California Western States, Sacramento, Calif.
Ralph T. Gilchrist, M. D.	Northwestern Mutual, Milwaukee, Wis.
Robert A. Goodell, M. D.	Phoenix Mutual, Hartford, Conn.
Harry W. Goos, M. D.	Home Life, Philadelphia, Pa.
J. Keith Gordon, M. D.	Sun Life Assurance, Montreal, Canada.
Angus S. Graham, Jr., M. D.	London Life, London, Canada.
LeRoy C. Grau, M. D.	Travelers, Hartford, Conn.
Robert J. Graves, M. D.	United Life & Accident, Concord, N. H.
Floyd M. Green, M. D.	Columbus Mutual, Columbus, Ohio
Frank L. Grosvenor, M. D.	Travelers, Hartford, Conn.

James R. Gudger, M. D.	Connecticut Mutual, Hartford, Conn.
Llewellyn Hall, M. D.	Phoenix Mutual, Hartford, Conn.
F. Tulley Hallam, M. D.	State Life, Indianapolis, Ind.
Frank Harnden, M. D.	Berkshire Life, Pittsfield, Mass.
Garland M. Harwood, M. D.	Life Ins. Co., Richmond, Va.
Walter C. Hausheer, M. D.	Prudential, Newark, N. J.
Harry M. Hawkins, M. D.	Old Line Life, Milwaukee, Wis.
Joseph K. P. Hawks, M. D.	State Farm Life, Bloomington, Ill.
William D. Heaton, M. D.	New York Life, New York City.
Ernest M. Henderson, M. D.	Confederation Life, Toronto, Canada.
Olin C. Hendrix, M. D.	New England Mutual, Boston, Mass.
Charles R. Henry, M. D.	Prov. Life & Acci., Chattanooga, Tenn.
Ivan C. Heron, M. D.	West Coast Life, San Francisco, Calif.
William L. Hilliard, M. D.	Equitable of Canada, Waterloo, Canada.
Daniel W. Hoare, M. D.	Penn Mutual, Philadelphia, Pa.
William W. Hobson, M. D.	Reliance Life, Pittsburgh, Pa.
Chauncey O. Hollinger, M. D.	Colonial Life, Jersey City, N. J.
Joseph C. Horan, M. D.	Metropolitan Life, New York City.
John L. Humphreys, M. D.	Reliance Life, Pittsburgh, Pa.
James H. Humphries, M. D.	Home Life, New York City.
J. Raymond B. Hutchinson, M. D.	Acacia Mutual, Washington, D. C.

Washington C. Huyler, M. D.	Mutual Life, New York City.
William G. Hyde, M. D.	Northwestern Mutual, Milwaukee, Wis.
Albert S. Irving, M. D.	American National, Galveston, Texas.
J. Grant Irving, M. D.	Aetna Life, Hartford, Conn.
Charles B. Irwin, M. D.	North American, Chicago, Ill.
Walter A. Jaquith, M. D.	Columbus Mutual, Columbus, Ohio.
Frank L. B. Jenney, M. D.	Federal Life, Chicago, Ill.
Albert O. Jimenis, M. D.	Metropolitan Life, New York City.
Albert E. Johann, M. D.	Bankers Life, Des Moines, Iowa.
William M. Jones, M. D.	Jefferson Standard, Greensboro, N. C.
George E. Kanouse, M. D.	Prudential, Newark, N. J.
Edward A. Keenleyside, M. D.	Connecticut General, Hartford, Conn.
Herbert B. Kennedy, M. D.	Woodmen of the World, Omaha, Neb.
Harry B. Kidd, M. D.	Metropolitan Life, New York City.
Oscar W. King, M. D.	Equitable Life Assurance, New York City.
Carl T. Kirchmaier, M. D.	Aetna Life, Hartford, Conn.
Henry B. Kirkland, M. D.	Prudential, Newark, N. J.
William P. Lamb, M. D.	Prudential, Newark, N. J.

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